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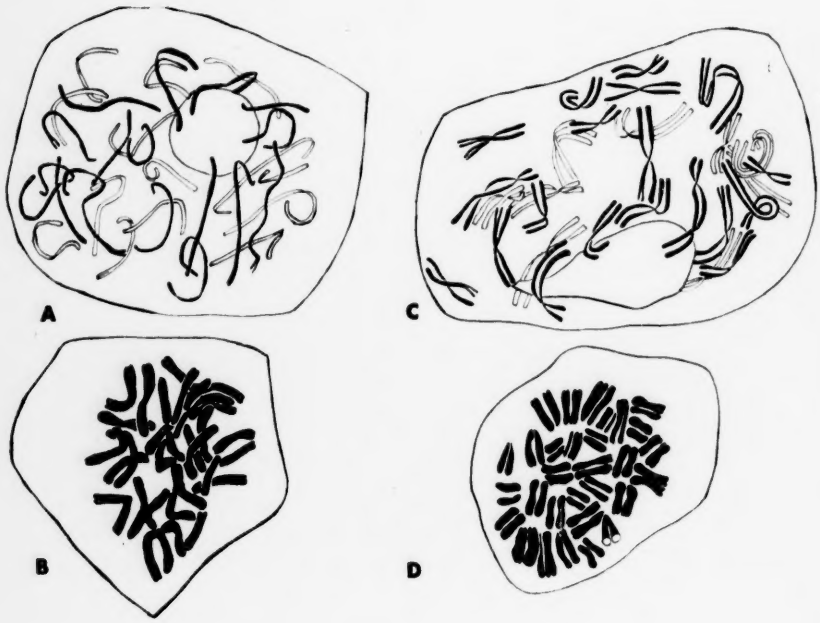

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Streaked Hairlessness in Cattle

Selection for Body-Weight in Mice

Eye-Color Mutants in the Honey Bee

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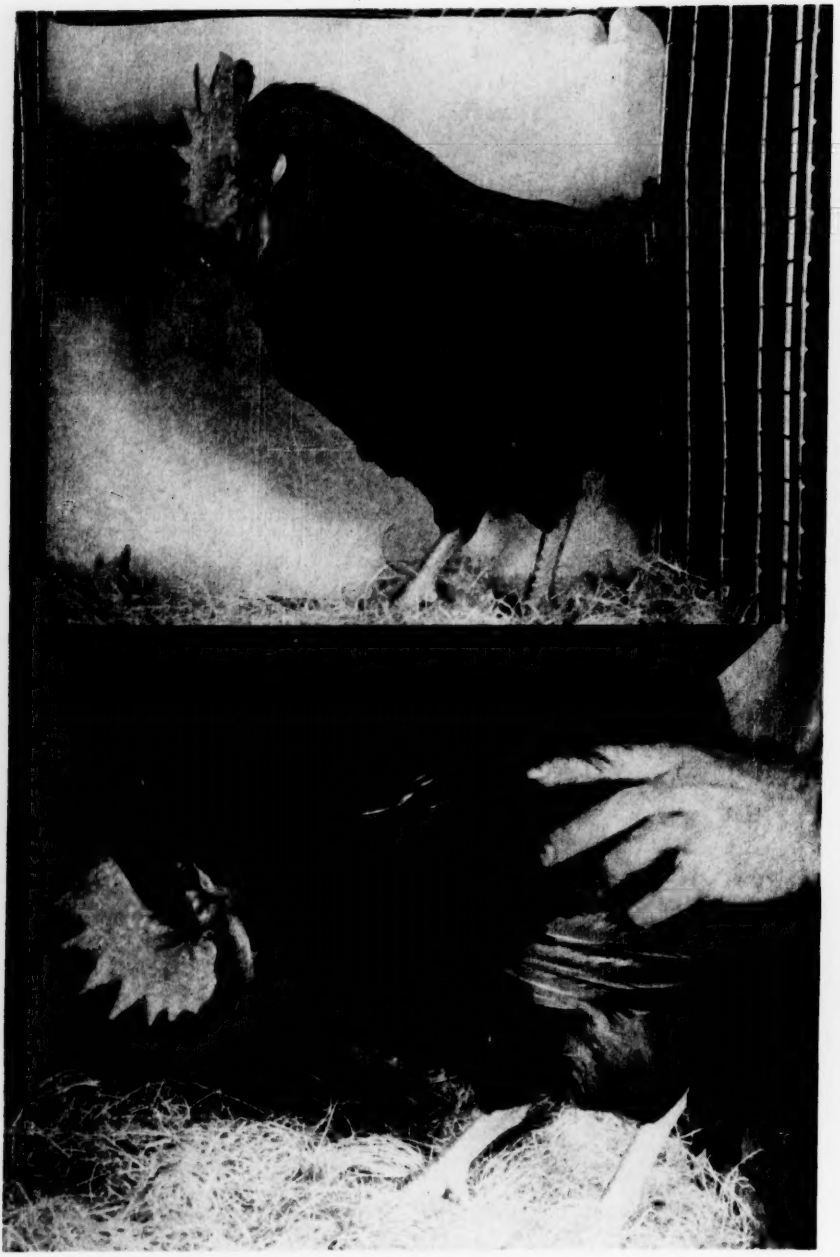

NEITHER PHILOSOPHER NOR BUDDIST—JUST A GENE

Figure 1

Appearance of the birds with the arched-neck condition. The typical stance of a bird with the arched-neck condition, when undisturbed, is shown above. Below is a bird as it appears in a state of excitement.

AN ARCHED-NECK CHARACTER IN CHICKENS

MARK H. CONNER AND C. S. SHAFFNER*

THERE have been several studies into the inheritance of anomalies of the head and neck in chickens. Jull and Quinn[2] reported on a "Crooked-Neck" character in 1931. They describe it as a twisted-neck defect, and say that it first appears when the birds are half-grown. An autosomal recessive gene is declared to be responsible. Scott, *et al.*[4] described a "Shaker" condition in White Leghorns. The affected bird moves the head and neck rapidly from side to side and walks with difficulty. The defect appears by the 18th day of age, and is said to be due to loss of cerebellar Purkinje cells. It is inherited by the action of a recessive sex-linked gene. "Jittery Chick" is another nervous phenomenon with a genetic basis, according to Bohren.[1] The affected birds retract their heads backwards and shake them rapidly. Bohren declares that the character is due to a recessive sex-linked gene. Knowlton[3] has described "Congenital Loco" as a condition in which the chick draws the head backwards and to one side, until it falls over. The birds die a few days after hatching. An autosomal recessive gene is responsible.

Methods and Results

The present study is concerned with the inheritance of an abnormal condition of the head and neck which appeared spontaneously in the University of Maryland flock of New Hampshire chickens. The F_1 and F_2 progenies were raised to four weeks of age in electrically heated batteries having screen-wire floors. They were then transferred to wire cages, each unit of which held three or four birds. At sexual maturity, the birds were put into individual laying cages. All of the afore-mentioned cages were housed in a concrete building under controlled heating and lighting conditions. Food and water were given *ad libitum*. The birds were fed University of Maryland starting mash for the first six to eight weeks, and then placed on a standard broiler mash.

Because the defect was a crippling one, making natural matings impossible, all matings were made by artificial insemination. A tuberculin syringe was used to deliver about 0.1 cc. of undiluted semen to each hen every two or three days.

Under the conditions of the experiment, the defect under study was never observed before the birds were 25 days old, and usually did not appear until they were two or three months of age. In a few instances, it appeared as late as seven months of age. It had the characteristics both of an anatomical fault and of a nervous disorder. The birds were normal in appearance when undisturbed except for a forward arching of the neck. When disturbed, the affected birds draw the head completely below the body and shake the head rapidly from side to side (Figure 1). This posture was usually maintained for some time, although often the birds fell backwards, apparently due to a loss of balance.

These symptoms bear some resemblance to those accompanying Newcastle disease in chickens. It had been noted that casual examination of affected chickens could lead to the erroneous conclusion that they had Newcastle disease, although none of the other external symptoms (respiratory difficulties, tremors, paralysis) was present. An investigation of possible genetic aspects of the defect was therefore thought to be in order.

The condition was first observed in one 5-month-old New Hampshire hen from the general farm flock. She was mated to her normal half brother (Table I). Of the nine chicks from this mating, five developed the condition by five months. This group of F_1 arched-necks was composed of three males and two females. Two other F_1 females became arched by seven months. Thus at this time the F_1 generation was composed of seven arched-neck birds (three males and four females), and two normals (one male and one female).

Three F_1 crosses were made: normal male $\times$ arched-neck female; arched-neck male $\times$ arched-neck female; arched-neck male $\times$ normal female. Eleven F_2 progeny were obtained, and have been observed to a maximum of seven months of age. Normal male $\times$ arched-neck female produced two normal females. Arched-

*Poultry Department, University of Maryland, College Park. Scientific Paper No. A421. Contribution No. 2454 of the Maryland Agricultural Experiment Station (Department of Poultry Husbandry).

neck male × arched-neck female gave four arched-neck males and two normal females. Time of onset of the condition in this group ranged from 25 days of age up to five months. Arched-neck male × normal female produced two normal females.

Discussion

The fertility of the birds was very low, and therefore the number of progeny in each generation was not great. It appears, however, that the character is not sex-linked, but is due to the action of several autosomal genes.

Semen-quality tests indicated that the reduced fertility encountered was due to the males. Initial motility, packed-cell volume, and cell count values were low for all F_1 and F_2 males tested, whether they exhibited the crooked-neck character or not. Methylene blue reduction time was correspondingly increased. Egg production and hatchability were not affected until the condition had become very severe.

Autopsies have shown that no internal lesions, such as those of Newcastle disease, leucosis, or nutritional deficiencies, were present in affected birds. No gross lesions of the central nervous system have been found. Death was usually due to an inability to eat and drink.

There is a variation in the severity of the condition. Some birds lived only a few weeks after onset, the condition becoming progressively more severe, while others have remained alive for as long as ten months. All the affected birds were thin and poor in appearance and undoubtedly would not survive under practical breeding conditions. Development of gross anatomical features other than those of the head and neck was normal.

Summary

Observations are presented on the inheritance of a new defect of the head and neck in New Hampshire chickens. The character is a crippling one and resembles the terminal symptoms of Newcastle disease in its appearance. It is inherited by the action of several autosomal genes.

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TABLE I. Matings Used and Progeny Obtained in Studying Inheritance of Arched-Neck Condition

Generation or mating	Birds		
P_1	N male × A female*		
F_1 progeny	3 A males; 1 N Male	4 A females; 1 N female	
F_1 crosses	N male × A female	A male × A female	A male × N female
F_2 progeny	2 N females	4 A males 2 N females	2 N females

* N = normal; A = arched neck.

(Received May 5, 1953)

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Note on the Silver Dapple Mutation of Shetland Ponies

MRS. John Jax of New York City has called my attention to a bit of evidence that the silver dapple mutation was already in existence in ponies in England prior to its identification in a strain of imported Shetlands in America as described by Castle and Smith in the July-August 1953 number of the Journal of Heredity.

The evidence is this. Lady Wentworth in her book *Thoroughbred Racing Stock*, London 1938, p. 56, prints a half-tone picture of "an improved type of highland pony, Skerryvore, owned by the late King Edward VII." This pony is dappled and has a light mane and tail, which are distinctive characteristics of the silver dapple variety as bred in America.

It should be noted in this connection that the Palomino mutation of horses, due to the dilution gene *D*, did not originate in the United States where its genetics has recently been investigated, but was brought to America centuries earlier from Spain by the conquistadores. It seems probable therefore the dominane mutant genes for both silver dapple and Palomino (possible alleles) have long been in existence, though largely overlooked until subjected to critical genetic study in recent times.

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THE INHERITANCE OF BLOOD TYPES IN THE DOG

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DURING the course of studying hemolytic diseases, Young and his group[1,5,10,11,12] at the University of Rochester prepared seven isoantibodies in dogs by the transfusion of incompatible blood.

Individual differences among dog bloods have long been known. In 1910 Von Dungern[8] used isoimmune sera to divide dogs into four groups based upon the reactions of two agglutinogens. Ottenberg, *et al.*[6] in 1913 demonstrated what appeared to be normally occurring isohemagglutinins and hemolysins in the dog. Although the reactions that occurred were generally weak and not easily repeated, they found that stronger isohemolysins could be developed by repeated transfusions of cells which were agglutinable by the recipient's normal serum. The repeated direct transfusion of animals having normally occurring isoantibodies with the whole blood of dogs which were apparently incompatible, gave rise to hemolytic reactions in the recipient.

Melnick *et al.*[3,4] found isohemagglutinins and isohemolysins in the serum of dogs following repeated injections of erythrocytes. The isoantibodies thus formed reacted with about 50 percent of the dogs tested. Dogs designated by them as "a" were capable of producing antibodies against cells from dogs designated as type "B." Dogs "B," however, did not produce antibodies in response to stimulation by cells "a."

Holman, *et al.*[2] as well as Wright,[9] observed occasional isoimmunization of recipient dogs as a result of antigenic stimulation of certain factors in the donors' cells. The hemolytic and agglutinative properties persisted in the serum for long periods of time and were intensified by renewed injections of the red cells.

In general, the observations of the isoimmunizations described so far were made during the course of physiological experiments in dogs. The work of the Rochester group is of greater significance to us, since they were interested in the isoantibodies and isoimmunization *per se*. The serologic and hematologic characteristics of five isoantibodies in the dog have been intensively studied and reported.[1,10,11,12] The antigenic factors defined by the antibodies are designated as A, B, C, D, and E in the order of their demonstration. The letters assigned carry no implication of any relationship between canine factors and the human blood groups designated by the same letters.

Sufficient numbers of dogs of known parentage for the satisfactory study of the heredity of dog blood types were not available to the Rochester group. For our study we had available at the dog colony of the Jackson Laboratory a large number of purebred dogs which had been used in the study of genetics and social behavior. Not only were relatively large numbers of dogs available, but in some cases three or four generations were on hand for study. The animals used included four generations of Basenjis, three generations of Cocker Spaniels, and five families of crosses between Basenjis and Cocker Spaniels.

Our original intention was the study of the relationship of dog breeds based on blood group frequencies, but it was seen very quickly that the random drift of the blood group frequencies in our

*The authors are indebted to Dr. William A. O'Brien, Jr. of the Department of Medicine, University of Rochester School of Medicine and Dentistry, Rochester, New York, for supplying the dog blood typing serum.

colony made it impossible to extrapolate our findings to include the whole of the breed.

The data reported here are actually from two different sources. The analysis of frequency of blood types and combinations of types and the mathematical tests of the hypotheses on the mode of inheritance are based upon data collected by the Rochester group. The data on actual breeding and inheritance were collected at the Jackson Laboratory, using the methods described by the Rochester group and the typing sera provided by them.

In our analysis of the inheritance of dog blood types, we were interested in possible parallelisms with the mode of inheritance of some of the human blood group systems: for example, as in the human ABO system, where there are three or more alleles at a single locus; or the MN type of inheritance of the MNS system, where both genes are detectable; or the P blood group, where P is inherited as a Mendelian dominant character and the recessive is not detectable. We did not test the possibility that a single allele might give rise to more than one blood group factor.

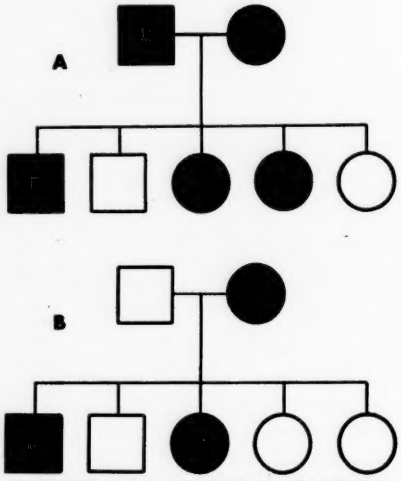
Table I shows the frequency of occurrence of four of the five different blood group factors in the Rochester group's random dogs, as well as results of tests of 124 dogs in our colony.

TABLE I. Frequency of occurrence of four of the five blood group factors

	Rochester Group	% (334 Tot.)	Jackson Colony	% (124 Tot.)	Combined	% (458)
A	204	59.5	101	81.4	305	66.6
B	23	6.7	16	8.87	39	8.5
C	326	94.5	124	100	450	98.2
D	67	19.4	38	30.64	105	22.9

TABLE II. Frequency of combinations of the four blood group factors

	Rochester Group		Jackson Colony		Combined	
Type	Frequency	%	Frequency	%	Frequency	%
ABCD	9	2.7	7	5.6	16	3.5
ABC	7	2.1	5	4.0	12	2.6
AC	155	46.5	58	46.6	213	46.5
ACD	28	8.4	31	25.0	59	12.8
BC	5	1.5	4	3.2	9	2.0
C	92	27.6	19	15.3	111	24.2
CD	29	8.7			29	6.3
AB	1	.3			1	.2
BCD	1	.3			1	.2
A	4	1.2			4	.9
None	3	.9			3	.7
	334		124		458	



INHERITANCE OF BLOOD FACTORS A AND D

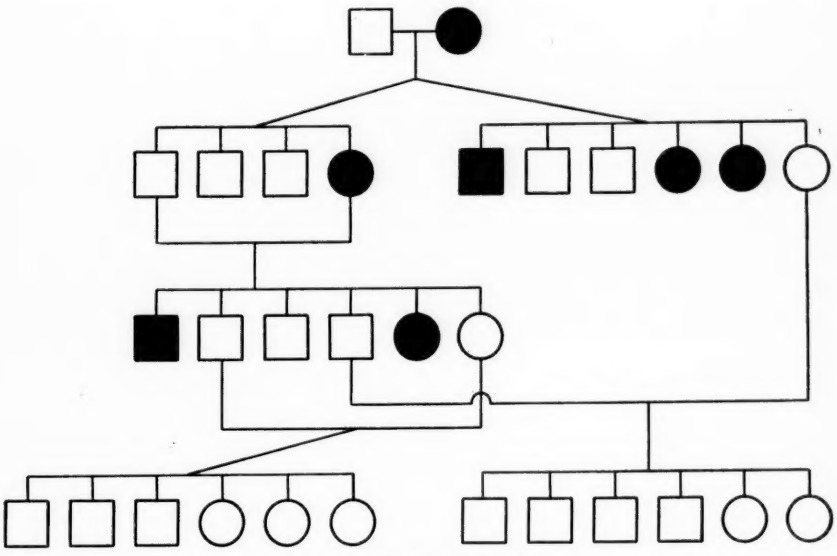
Figure 2

A—The inheritance of blood group factor A in a family of Cocker Spaniels; *B*—blood group factor D in a cross between Basenji and Cocker Spaniel.

Table II shows the frequency of combinations of the four blood group factors.

Since there were 16 dogs with type ABCD, it is evident that there are no more than two detectable genes responsible for blood group factors at any one locus; for if there were three detectable alleles, not more than two would appear in one individual, and the presence of two members of the triad would mean the exclusion of the third. As a corollary to this deduction it may be stated that at least three loci must be involved.

If any of the genes for the different types are alleles, the sum of their frequencies in the population should be greater than one. For example, consider the genes for the MN antigens of the human MNS system: the gene for M might occur in 75 percent of the population, and the gene for N in 75 percent. One-half of the population will have both genes. In a hypothetical system, then, gene X might occur in 75 percent of the population and allele X^p in 75 percent. Here one-half of the population will have both genes. If both X and X^p give rise to detectable antigens, the situation is fairly simple. Under this assumption, and substituting the observed frequencies of the various blood groups for X and X^p, we can rule out the following as allelic pairs: *AB, AD, BD.*



DOMINANT INHERITANCE

Figure 3

The inheritance of blood group factor B in a family of Basenjis.

Possible genetic systems must include multiple allelic series. Table III shows a hypothetical genetic system based on the possibility that genes X^x, X^y, and X^o yield two detectable blood groups, X and Y, but that there is no test for O. Thus we cannot differentiate between all genetic classes. This is parallel to what occurs in the human ABO system.

Table IV gives the blood types resulting from some matings which occurred in our dog population. Lines 1 and 2 give evidence that factors A and B are not due to a multiple allelic series, since we obtain animals which have neither factor A nor B from crosses involving AB by non-AB. Similarly, B and D can be shown to be non-alleles by the matings indicated on line 3. In our studies to date we have not had the proper matings to look for the segregation of A and D. However, the Basenjis in our colony are all homozygous A and show segregation of D. For corroborating evidence we must therefore rely upon the test of this genetic system which has been carried out by calculating the expected values of the frequency of the XY group using the method devised by Fisher, which is shown by Race and Sanger.[7] The results of this determination are summarized in Table V. If we assume that the dogs studied by the Rochester group are the results of random mating, the hypothesis that these factors occur as allelic groups can be rejected with a high degree of confidence.

A series of pedigrees illustrating the inheritance of blood group factors *A*, *B*, and *D*, based upon results of testing animals at the Jackson Laboratory colony, is shown in Fig-

TABLE III. Hypothetical genetic system for blood types based upon three alleles*

Genotype	Serological Type
X^xX^x X^xX^o	X
X^xX^y	XY
X^yX^y X^yX^o	Y
X^oX^o	Neither X nor Y

* X^y refers to gene; Y refers to antigen

TABLE IV. Blood types of parents and offspring involved in some matings at the Jackson Laboratory colony

Parental types	Number of litters	— Types of Offspring — ABC	AC	BC	C	ABCD	ACD
1) ABC × C	3		4	2	4		
2) ABC × AC	2	1	5	2	2		
3) ABCD × AC	3	1	4			4	7

ures 2 and 3. Factor C is present in all our animals and thus need not be shown.

We can safely state, then, that the blood group factors in dogs are inherited as simple Mendelian dominants. There is no evidence as yet of linkage or multiple allelic series.

As an interesting sidelight to the blood typing in the Basenjis, we found that factor B did not come down to the latest generation of Basenjis. Our typing showed that in a group of random matings in Basenjis, only animals of type AC or ADC had been included. As a result, factor B was lost. The loss of this factor indicates quite clearly how chance mating in a small population may give rise ultimately to vastly different blood group frequencies from those which occur in other populations. This is shown clearly in the pedigrees in Figure 2.

Summary

The frequency of occurrence of blood types in dogs was analyzed. The results show that according to the systems studied above, the factors appear to be inherited as simple Mendelian dominants with no evidence of linkage or multiple allelic pairs.

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TABLE V. Test of the hypothetical genetic system for blood types based upon three alleles

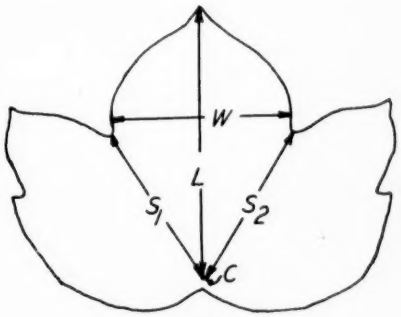
		Observed Serological Types				Expected	Deviation	Variance	χ^2
	Class	XY	X	Y	O	XY			d.f. = 1
1	AB	17	187	6	124	3.41	13.59	5.43	34.01
2	BC	22	1	304	7	4.84	17.16	30.79	9.56
3	AC	199	5	127	3	21.25	177.75	102.92	306.98
4	AD	37	167	30	100	15.10	21.8	25.66	18.53
5	BD	10	13	57	254	1.5	8.5	1.69	42.75
6	CD	67	259	0	8	32.4	99.4	164.87	59.93
7	O				3				

(*Received August 27, 1953*)

SUB OKRA, A NEW LEAF SHAPE IN UPLAND COTTON

JOHN M. GREEN*

AN unusual leaf shape in upland cotton (*Gossypium hirsutum* L.) observed in the breeding plots of the Coker's Pedigreed Seed Company of Hartsville, South Carolina, was obtained from that company by Dr. Thomas Kerr of the United States Department of Agriculture. The material in which this leaf shape was found was derived from hybrids involving an amphidiploid synthesized from *G. arboreum* and *G. thurberi* by the late J. O. Beasley. The possibility that its unusual leaf shape resulted from a gene or genes transferred from one or both of the diploid species was immediately recognized. As a preliminary step in the study of this new leaf shape, its inheritance and relationship to other leaf shapes in upland cotton have been investigated.



A NEW LEAF SHAPE
Figure 4

Diagrammatic outline of normal broad leaf of upland cotton showing the measurements used in characterizing the leaf types worked with. W was taken at the widest portion of the central lobe. S, as used in the text, refers to the mean value of S_1 and S_2. C is the center of the callus spot.

Materials and Methods

A series of alleles for normal (broad), okra, and super okra leaf shapes has been established in *G. hirsutum*.[2,5] Hutchinson and Silow[3] have recommended the symbols *l*, L^o, and L^s, respectively, for the above leaf types. Another leaf shape which has been found to be independent of the okra series is the recessive round leaf.[1,4] It was given the symbol *rl* by Brown and Cotton.[1] For the purpose of convenience of expression in presentation of the results of this study, the conclusion reached will be anticipated and the name suggested for the new leaf shape, sub okra, and the accompanying symbol L^{so}, will be used throughout the remainder of this paper.

Crosses of sub okra × okra and sub okra × round were made in the field in 1948. The F_1 hybrids were backcrossed to sub okra and outcrossed to normal broad leaf in the greenhouse. The cross sub okra × broad was also made in the greenhouse. All parents, F_1's, and backcross and outcross progenies were grown in space-planted rows in the field in 1949. A small F_2 population of the sub okra × round cross was also grown.

Visual classifications for leaf shape were verified by measurements made on primary leaves. Dimensions measured were those described by Stephens.[6] These are illustrated in Figure 4. Stephens considered that "under good growing conditions the climax leaf is produced at the onset of flowering."

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OKRA LEAF SHAPES

Figure 5

Leaf shapes produced by the four alleles of the "okra series"; upper left, broad (l); upper right, sub okra (L^u); lower left, okra (L^o); lower right, super okra (L^s). The sub okra gene is believed to be derived from *Gossypium thurberi*.

This writer found that primary leaves at the 8th to the 12th nodes measured after the plants had formed 16 or more nodes and after flowering had begun gave uniform results adequate for purposes of classification. In the parents and F_1 hybrids, three leaves on each of three plants were measured. In the segregating progenies, of course, single plant determinations had to be used for classification purposes. Differences were found to be sufficiently distinct and consistent so that no refined statistical approach was necessary. Not all plants classified were measured; only enough were measured to verify the validity of visual classification.

Experimental Results

Characteristics of the leaves of the parents and the F_1 hybrids are given numerically in Table I. The data indicate distinct differences among the parents and that the F_1 hybrids were intermediate between the parents in relative width of the central lobe and in degree of laciniation. In verification of visual classifications the ratio W/(L-S) was used, since it

TABLE I. Characteristics of the leaves of the parent strains and F_1 hybrids expressed numerically as various indices of leaf shape

	Genotype						
Ratio	ll	L^ul	L^ol	L^uL^u	L^uL^o	L^oL^o	L^sL^s
S/L	.546	.341	.272	.217	.197	.105	.011
S/W	.865	.659	.667	.468	.482	.383	.059
W/L	.631	.518	.407	.486	.408	.274	.181
W/(L-S)	1.396	.787	.559	.622	.508	.310	.183

took into account both relative width and laciniation.

The F_1 of the sub okra × round cross was very similar in appearance to the sub okra × broad F_1. In the backcross (round × sub okra) × sub okra, two classes, sub okra and intermediate sub okra / broad, were readily distinguished. These approached a 1:1 ratio, P = .5 - .7. Corroborating results were obtained in the outcross to broad, where a ratio of one intermediate sub okra / broad: one broad was approximated, P= .5 - .7 (Table II). Since round was recessive, it did not appear in either the backcross or the outcross.

Six classes, sub okra, intermediate sub okra/ broad, broad, sub okra / round, intermediate sub okra / round, and broad / round, were observed in a small F_2 progeny of the cross round × sub okra. Because of the small size of the population, the last three classes were combined. Phenotypes, genotypes, and expected and observed frequencies are presented in Table III. The close agreement of observed and expected support the interpretation given, and it seems evident that sub okra and round are neither allelomorphic nor linked. Because the round gene was apparently completely recessive, the round × sub okra cross was equivalent to a broad × sub okra cross. Evidence that sub okra and broad differ by a single gene found in the results of this cross seems to the writer to be conclusive.

Results of the sub okra × okra backcross and outcross are given in Table IV. In each case only two phenotypic classes were observed, indicating that sub okra and okra are allelomorphic. While in the outcross the fit to a 1:1 ratio is not especially good, P = .05 - .10, the fact that only two classes were observed is considered to be good evidence in support of the hypothesis.

Discussion

The results presented indicate that the new leaf shape studied is a member of the progressive series of alleles: broad, Sea Island, okra, sub okra, and super okra. Leaves of all these types, except Sea Island, are shown in Figure 5. It is evident from this photograph and from the data of Table I that there is a progressive reduction in the lobe width and a progressive increase in the depth of the laciniae from the broad through the super okra shapes. In view of the regularity of this series and in conformity with the names okra and super okra, the name sub okra is suggested for the new member of the series. The gene symbol suggested is L^u. Since both "*S*" and "*B*" have been used for superscripts for leaf shape gene symbols, the middle letter of "sub" is suggested.

Independent evidence indicates that the sub okra gene was derived from *G. thurberi*. This would place the series of leaf shape genes in the D (or Wild American) subgenom of upland cotton. This is contrary to the concept advanced by Stephens[8] who postulated that the New World cottons had two leaf shape loci and that the allelomorphic series in question could be considered as a series of Asiatic alleles.

The sub okra leaf shape bears a striking resemblance to the Sea Island shape transferred to an upland background as drawn by Stephens.[6] That this shape (L^s) is a member of the okra series in upland was concluded by Stephens.[7]

Summary

A new leaf shape in upland cotton was found to be inherited simply and to be a member of the leaf shape series l-L^u-L^o-L^+. The name sub okra and the gene symbol L^u are suggested for identifying this character.

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TABLE II. Observed frequencies in the backcross and outcross progenies of the cross round × sub okra

Cross	Genotypes		Total	χ^2	P
	L^uL^u	L^ul			
$(L^u \times l) \times L^u$	68	76	144	.444	.5 - .7
	L^ul	ll			
$(L^u \times l) \times l$	32	37	69	.362	.5 - .7

TABLE IV. Observed frequencies in the backcross and outcross progenies of the cross sub okra × okra

Cross	Genotypes		Total	χ^2	P
	L^uL^u	L^uL^o			
$(L^u \times L^o) \times L^u$	97	96	193	0.005	.9 - .95
	L^ol	L^ul			
$(L^u \times L^o) \times l$	107	84	191	2.246	.05 - .10

TABLE III. Observed and expected frequencies in the F_2 progeny of the cross round × sub okra

Phenotype	Genotype	Ratio	Frequencies	
			Expected	Observed
Sub okra	L^uL^u Rl Rl L^uL^u Rl rl	3	12.2	9
Intermediate sub okra/broad	L^ul Rl Rl L^ul Rl rl	6	24.3	29
Broad	ll Rl Rl ll Rl rl	3	12.2	11
Round	L^uL^u rl rl L^ul rl rl ll rl rl	4	16.3	16
Totals		16	65.0	65

$\chi^2 = 1.872$ P = .5 - .7

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(*Received September 21, 1953*)

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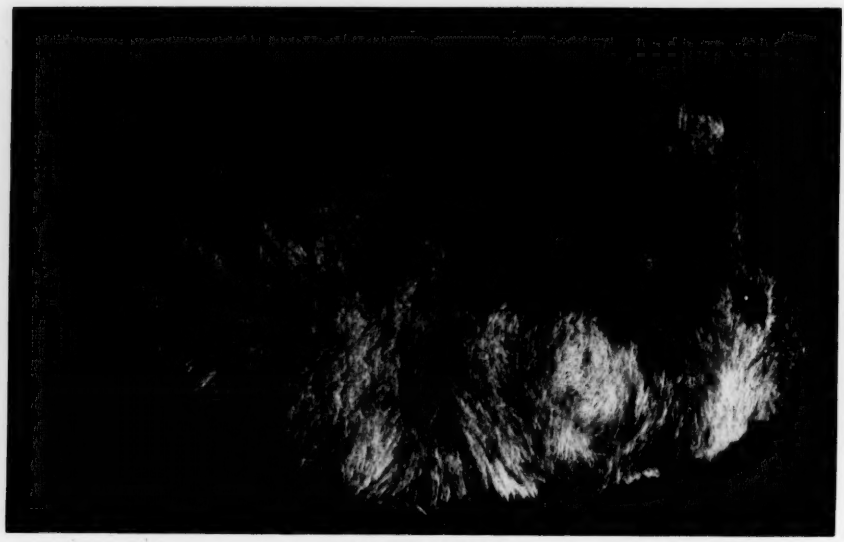
"Fuzzy," a Dominant Mutation in the Guinea Pig



WAS HE FUZZY!
Figure 6
The fuzziness is evident at birth and becomes more pronounced with age.

Two fuzzy haired guinea pigs, a male and a female, were noted in a litter from presumably normal parents of the Naval Biological Laboratory (NBL) strain. Although the rest of the litter and the parents were not available for examination, there is little doubt that they were normal since fuzzy animals would have been easily identified.

The NBL strain was developed in this laboratory from a breeding nucleus of 16 females and four males and has been maintained by pen breeding as a closed colony since 1945. No morphological or physiological mutations, excepting the fuzzy mutation, have been noted. Although the strain continues to segregate for both color and coat patterns, it has been relatively uniform in response to graded infections and has been extensively used for studies in experimental epidemiology.

Fuzzy guinea pigs may be identified at birth by their curly vibrissae. The fuzziness becomes apparent within two to three days and grows more pronounced with time (Figure 6).

The original fuzzy guinea pigs when mated with normal animals yielded both fuzzy and normal progeny. Only fuzzy offspring were observed in litters from fuzzy parents; the number of guinea pigs from such matings was too small to expect the 25 percent normal guinea pigs from matings between heterozygous parents. These data suggest the original fuzzy guinea pigs were heterozygous for a dominant gene which has been given the symbol *Fz*.

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EFFECTIVENESS OF SELECTION FOR BODY WEIGHT IN MICE

From Inbred and Outbred Populations Derived from Common Parent Stocks

W. L. Lewis and E. J. Warwick*

THE effectiveness of selection in changing gene frequencies and thereby changing the population means has been amply demonstrated for certain characteristics. Selection for high and low fat and high and low protein content in corn at the Illinois Agricultural Experiment Station (Winter[10]) was highly successful in changing the means, but variation did not decrease. In mice, Goodale[4] reported an increase of about 40 percent in body weight after seven generations of selection involving the use of the progeny test. MacArthur[7] selected mice for high and low body weight for 25 generations, using individual and some progeny selection without deliberate inbreeding. The two group averages diverged about 24 grams, thus demonstrating the effectiveness of selection. Progress was becoming slower in later generations of selection and Butler[2] who continued selection in MacArthur's stocks through the 32nd generation, doubts that any real progress was made after the 21st generation. There was some evidence of actual reversal of progress in later generations. Size was the only trait for which deliberate selection was practiced, but certain correlated characters including color, docility, tendency toward obesity, and litter size became typical for each line.

Mather and Harrison[8, 9] selected *Drosophila melanogaster* for high and low numbers of abdominal chaetae. Selection was effective more or less intermittently, but progress was usually accompanied by lowered fertility which resulted in the extinction of most lines by the 20th to 35th generation of selection.

Recent studies of the effectiveness of selection in swine (summarized by Dickerson *et al.*[3]) have indicated extremely low effectiveness of selection for litter size, litter weaning weight, and body weight at weaning and 154 days of age in swine populations in process of inbreeding.

The present study with mice was undertaken to compare the effect of selection for large, medium, and small body size in inbred and outbred populations upon both the averages and the variability.

Materials and Methods

Formation and Propagation of Stocks:

The foundation stocks for this experiment were furnished in February, 1948, by the late Dr. J. W. MacArthur then of Toronto University. They consisted of from 10 to 12 males and 10 to 15 females each of (1) a stock selected for large body size for 25 generations, (2) a stock selected for small body size for 25 generations, and (3) an unselected random bred stock of medium size descending from the same foundation as the two selections. (See MacArthur,[7] for information on these stocks.)

While deliberate inbreeding had not been practiced, the population size made it probable that inbreeding had increased at the rate of one to three percent per generation. Thus, the amount of genetic variability had probably been greatly reduced. In an attempt to recover as much of the original variability as possible and at the same time maintain stocks of different sizes for the crossing tests which were conducted simultaneously, the large and small size groups were each crossed with the unselected, random-bred stock of average size.

The progeny of four† males of the random-

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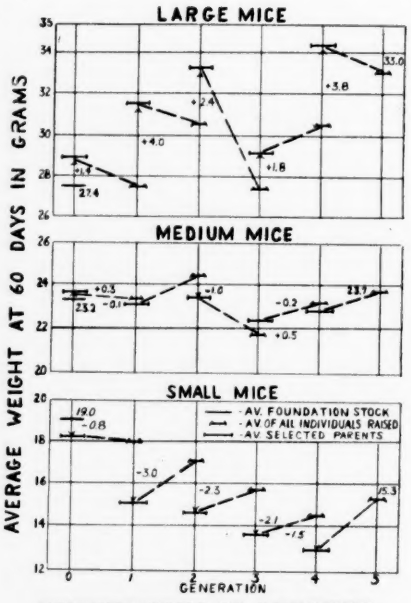
†Originally five sires were used in each group, but it early became apparent that for the limited facilities the resulting populations would be too large; so all progeny of one sire in each group were eliminated.

bred group crossed with 42 small females formed the foundation stock upon which selection for small size was practiced. A few females from the reciprocal cross were also used. This stock is hereafter referred to as "small." Similarly, the progeny of four males of the random-bred group crossed with 37 females of the large group (plus a few females from the reciprocal cross as needed to make up numbers) formed the foundation stock upon which selection for large size was practiced and is hereafter referred to as "large." Four average sized males of the unselected random-bred stock were bred to females of the same group to produce the foundation stock upon which selection for average size was practiced. These are referred to in this paper as "mediums."

Among the foundation stocks of each size group one son (plus a full brother to serve as a reserve and be used as necessary) of each male in the previous generation was selected for adherance to the size for which selection was made. Virtually all available foundation females were used. Attempts were made to mate each selected male with 10 half or full sisters and 10 unrelated females. In most cases the reserve male also had to be used to accomplish this, but in either case half of the resulting litters were inbred and half outbred. The inbred progeny of each sire (or of his reserve) were thereafter propagated as a closed line ideally composed of a single male and 10 females per generation. In practice, two (or in rare cases three) males sometimes had to be used if the one originally selected proved to be of low fertility. In some cases sterility among the females resulted in fewer than 10 litters being raised. No lines were lost, however, by reason of sterility.

The outbred progeny of each foundation sire (or of his reserve) went into an outbred group which in each size group was thereafter ideally composed of four males and 40 females per generation. As in the inbreds, low fertility or sterility sometimes resulted in more males being used or in fewer than 40 litters being raised.

Only one litter per female was raised, and males were used only on contemporary females. Selection of breeding animals for the next generation in the inbred lines was from within the line. In the outbreds, one male and 10 female progeny of each sire (or of his reserve) used in the previous generation were selected. Selection was thus between individuals within sire progenies, but no between-sire selection was practiced. Matings in the so-called outbred population of each size group were made between sire progenies according to a definite plan to keep inbreeding to a minimum. In the third, fourth, and fifth generation, this system unavoidably involved matings between animals related on the average somewhat less than quadruple second cousins and therefore resulted in a low degree of inbreeding. Individual inbreeding coefficients have not been calculated for these outbred populations but according to Wright[11] a breeding system of this kind would result in average increases in inbreeding of less than 2½ percent per generation. Thus, the average inbreeding in the fifth generation probably did not exceed seven percent. The occasional use of a reserve male should have reduced this somewhat. The coefficients of inbreeding for each inbred line were calculated after the method of Wright.[11]



EFFECTIVENESS OF SELECTION

Figure 7

The average weight at 60 days for the three size groups is shown graphically. The effectiveness of selection varied considerably from generation to generation.

The procedures followed in forming and propagating the inbred and outbred stocks should have (1) given initially comparable groups to work with since inbreds and outbreds came from a common parentage, and (2) permitted selection of equal intensity within the limits imposed by reproductive rates.

Selection Procedures and Management:

Selection in the large and small groups was for body weight at 60 days of age. Selection in the medium group was for males weighing 25 grams and females weighing 21 grams at 60 days, since this was the average weight of the foundation stock. Selection was entirely for individual body weight with the restrictions that (1) no animals were selected from litters

of four or less raised, and (2) no more than three females were saved from any one litter. Occasionally, these procedures had to be modified if numbers of individuals or litters raised were exceptionally low in a group. In several of the small lines a number of dwarfs or pygmies weighing less than 10 grams at 60 days of age were reared. These were completely sterile and could not be used for breeding.

Females were mated in groups but removed to individual cages for littering. All litters of over 10 were reduced to 10 the first day. At weaning (21 days) the mice were sexed, individually marked, weighed, and caged in groups according to age, sex and group. They were weighed again at 60 days of age.

The mice were housed in a small animal house which was not air-conditioned. Uniform temperatures were maintained in the winter by a thermostatically controlled heating system. Occasional high summer temperatures apparently resulted in slower gains and some death losses. The seasonal changes, although erratic at times, were shared by all groups. Each generation was contemporary for all groups. The mice were fed Purina laboratory chow and water *ad libitum*.

Summary and Analysis of Data:

For analysis, the average weight of a litter was considered to be the unweighted average of the separate averages of the male and the female mice in the litter. In case a litter included mice of only one sex, the average was adjusted upward or downward according to the average difference between the two sexes in the size groups concerned. The averages given by sizes or breeding groups are unweighted averages of the litter means. Statements regarding the statistical significance of differences in 60-day weights are based on analysis of variance of the unweighted litter means.

Results and Discussion

Trends in Body Weight, Litter Size, and Correlated Characters:

Small Size Group. As summarized in Table I, the average number of mice raised per litter was smaller in the inbreds than in the outbreds from the first through the fifth generation. A gradual downward trend was observed in weaning weight (21 days) with no consistent differences being observed between breeding

TABLE I. Summary of the Production Data

Generation	Group	Av. Fx	No. Litters	Av. No. Raised Per Litter	Av. Wts. (grams)* 21 da.	60 da.
SMALL						
Foundation		0	42	5.6	9.2	19.0
First	Inbred	15	40	7.2	9.2	17.8
	Outbred	0	40	8.0	9.0	17.8
Second	Inbred	22	40	5.6	8.0	16.7
	Outbred	0	41	6.1	8.0	16.2
Third	Inbred	31	44	4.7	7.4	14.4
	Outbred	slight	43	5.1	7.8	14.5
Fourth	Inbred	37	41	5.1	7.2	13.6
	Outbred	slight	40	5.5	7.0	13.1
Fifth	Inbred	42	35	4.6	7.4	13.1
	Outbred	4-6†	40	5.5	7.3	13.7
MEDIUM						
Foundation		0	32	7.4	10.0	23.2
First	Inbred	15	37	7.7	10.8	23.7
	Outbred	0	36	8.3	9.8	22.9
Second	Inbred	22	40	7.8	10.4	24.0
	Outbred	0	41	6.8	10.6	24.5
Third	Inbred	31	40	7.0	9.1	21.6
	Outbred	slight	37	6.5	9.6	21.8
Fourth	Inbred	37	41	7.1	9.0	22.8
	Outbred	slight	40	7.6	9.6	23.1
Fifth	Inbred	42	37	6.5	9.2	23.4
	Outbred	4-6†	42	6.4	10.6	24.0
LARGE						
Foundation		0	32	8.4	9.2	27.4
First	Inbred	16	40	8.2	11.7	27.4
	Outbred	0	41	8.6	11.6	27.4
Second	Inbred	19	42	7.3	11.8	31.2
	Outbred	0	40	7.9	12.4	29.9
Third	Inbred	33	28	3.2	10.9	28.6
	Outbred	slight	39	5.1	8.7	26.6
Fourth	Inbred	39	36	6.9	10.8	31.0
	Outbred	slight	43	7.9	10.6	30.0
Fifth	Inbred	44	36	5.1	11.3	31.8
	Outbred	4-6†	40	6.3	12.6	33.9

*Unweighted average of average male and female weights.
†Estimated.

TABLE II. Mean Superiority or Inferiority of Selected Parents, at 60 Days of Age (grams) in the Small, Medium, and Large Size Groups*

Generation of Selection	Small			Medium			Large		
	Wtd. Av. of 4 Inbred Lines	Wtd. Av. of Outbred Groups	Wtd. Av. Inbred and Outbred Combined	Wtd. Av. of 4 Inbred Lines	Wtd. Av. of Outbred Groups	Wtd. Av. Inbred and Outbred Combined	Wtd. Av. of 4 Inbred Lines	Wtd. Av. of Outbred Groups	Wtd. Av. Inbred and Outbred Combined
All zero gen. individuals	19.0	19.0	19.0	23.2	23.2	23.2	27.4	27.4	27.4
Selected parents	17.8	18.6	18.2	23.6	23.4	23.5	29.0	28.7	28.8
Selection differential	—1.2	—0.4	—0.8	+0.4	+0.2	+0.3	+1.6	+1.3	+1.4
All 1st gen. individuals	17.7	18.2	18.0	24.0	22.9	23.3	27.3	27.4	27.4
Selected parents	15.2	14.8	15.0	23.5	22.7	23.2	32.0	31.0	31.4
Selection differential	—2.5	—3.4	—3.2	—0.5	—0.2	—0.1	+4.7	+3.6	+4.0
All 2nd gen. individuals	17.4	16.4	16.9	24.0	24.5	24.2	31.2	29.9	30.6
Selected parents	15.3	13.9	14.6	23.2	23.3	23.2	33.5	32.6	33.0
Selection differential	—2.1	—2.5	—2.3	—0.8	—1.2	—1.0	+2.3	+2.7	+2.4
All 3rd gen. individuals	16.1	15.2	15.6	21.6	21.8	21.7	28.6	26.6	27.2
Selected parents	14.4	12.6	13.5	22.0	22.4	22.2	29.3	28.8	29.0
Selection differential	—1.7	—2.6	—2.1	+0.4	+0.6	+0.5	+0.7	+2.2	+1.8
All 4th gen. individuals	14.3	14.7	14.5	22.8	23.1	23.0	31.0	30.0	30.4
Selected parents	13.2	12.9	13.0	22.7	23.0	22.8	33.8	34.6	34.2
Selection differential	—1.1	—1.8	—1.5	—0.1	—0.1	—0.2	+2.8	+4.6	+3.8
All 5th gen. individuals	15.7	15.0	15.3	23.4	24.0	23.7	31.8	33.9	33.0

*Dwarf mice have been omitted from these calculations. Thus, the averages given by generations are for normal mice and the selection differentials are the differences between selected individuals and the normal mice of their generation.

types. A downward trend, presumably resulting from selection, was observed in 60-day weight with the outbreds being consistently but not significantly lighter than the inbreds. Regression coefficients of mean weight on generation were significantly negative in both inbreds and outbreds and did not differ significantly. In no generation was the difference between 60-day weights of inbreds and outbreds significantly different.

Medium Size Group. There was a gradual decline in litter size from generation to generation in both breeding types. Litter sizes of inbreds and outbreds did not differ consistently. The inbreds were slightly smaller at weaning than the outbreds from the second through the fifth generation, but the differences were not statistically significant. The average 60-day weight from generation to generation tended to fluctuate around the mean of the original random-bred population (males 25 grams, females 21 grams).

Large Size Group. A gradual decline in the number of mice raised was observed in both breeding types with the decline being consistently greater in the inbreds than in the outbreds (see Table I). Weaning weight fluctuated somewhat from generation to generation with no evidence of a trend in either breeding group. There was an upward trend in 60-day weight with the inbreds being as heavy or heavier than the outbreds in each generation except the fifth. Regression coefficients of mean 60-day weight on generation were significantly positive in both inbreds and outbreds and did not differ significantly. The suggestion is strong that inbreeding had very little depressing effect on 60-day weight.

Correlated Characters.

Our observations confirm those of MacArthur[7] in indicating that the mice selected for small size tend to be active, to have small litters, and to be predominantly black in color. The large groups tend to be obese, sluggish, and brown or albino in color. A gene for dwarfism was apparently present in low frequency in the original small stock, but increased in frequency as selection progressed. This is discussed in a separate paper.

Amount and Effectiveness of Selection Practiced.

The selection differentials for 60-day weight in the small (omitting dwarfs form calculations), medium, and large size groups by generation and breeding type are given in Table II. The selection differentials for inbreds and outbreds have been combined with each of the three size groups and are presented graphically with mean body weights per generation in Figure 7.

Heritables calculated by dividing the additional divergence built up between large and small groups during the period being considered by the cumulative selection differentials during the same period are given in Table III.

Over all generations the heritabilities were 42 percent and 37 percent, respectively, for the outbreds and the inbreds. According to Lush[6] the estimation of heritability by this method will include a considerable amount of epistatic variation as well as the amount due to genes with additive effects in the first two or three generations while in later generations the heritability estimate will include primarily additive effects. As can be seen from Figure 7, the effectiveness of selection varied considerably from generation to generation. Since the foundation stocks were crosses between MacArthur's original large and small and medium groups, it is possible that the high apparent effectiveness of selection in early generations resulted from selection for a few genes with major effects.

Whatever the reason, selection became less effective in generations two to five as indicated by heritability estimates of 33 and 21 percent for the outbred and inbred groups, respectively.

The lower average heritability for the inbreds comes almost entirely from differences in effectiveness of selection in the last generation. From the fixation of genes taking place with inbreeding, it would be expected that selection in the inbreds would be 21 percent less effective over the entire five generations and 30 percent less effective over the last three generations. Actual reductions in heritability for the two periods were 12 percent and 35 percent less effective, respectively. Therefore there seems no reason to suspect from these data that inbreeding had a depressing effect on heritability greater than expected from its effect on homozygosity.

As shown in Table IV, variability increased with selection in the small mice when measured by either the standard deviation or coefficient of variation. This increase was due to the occurrence of dwarfs in later generations. When they are excluded, the variability increases only a little.

The coefficient of variation in the medium and large size groups did not change appreciably with selection. In no size group was there conclusive evidence of a reduction of variability in the inbreds as compared to outbreds although in the last two generations in each size group the inbreds were slightly less variable than outbreds.

The results of this experiment are consistent with others in indicating a negligible effect of selection on uniformity.

Except for the fact that no breeding animals were selected from litters of less than four, there was no intentional selection for litter size. Due to the fact that larger litters have more individuals available than smaller ones, there was automatic selection for litter size. Detailed analyses not given here showed an overall selection from litters of about 1.1 mice above the average. Of this only about .1 was intentional, the remainder being automatic.

However, in spite of this selection, average litter size born and raised decreased in all groups. Whether this was due to some undetected factor in the environment or to the indirect effects of selection for body size is unknown. Mather and Harrison[8, 9] observed detrimental effects on fertility from both plus and minus selection for chaeta number in *Drosophila*.

Occurrence of Dwarfism.

No dwarfs were born in the foundation stocks and in the first generation of selection only one dwarf was produced. In the next generation, dwarfs were produced in the small outbred population and in three of the four small inbred lines. No dwarfs were ever produced in the other small inbred line or in any of the medium or large groups.

The frequency of dwarfs increased to the third generation and thereafter tended to remain at a high level. In the groups with dwarfism 15.5 percent of the 1229 mice raised

TABLE III. Heritability of Weight at 60 Days of Age Estimated from the Results of Selection

Item	Generation	Estimate of Heritability (percent)
Inbred lines (large and small)	all generations	37.2
	2-5 generations only	21.5
Outbred groups	all generations	42.0
	2-5 generations only	33.0
Inbred lines and Outbred groups combined	all generations	40.2
	2-5 generations only	28.8

TABLE IV. Means, Intra-group Standard Deviations, and Coefficients of Variability for 60-Day Weights by Generations

	Inbreds			Outbreds		
Size and Generation	Av. 60-day Wt.	St. Dev.*	C.V.	Av. 60-day Wt.	St. Dev.*	C.V.
ALL SMALLS						
Gen. 1	17.8	2.30	12.9	18.1	2.28	12.5
Gen. 2	16.7	3.36	20.1	16.2	2.78	17.1
Gen. 3	14.4	4.20	29.1	14.5	2.84	19.5
Gen. 4	13.6	2.87	21.1	13.1	3.79	28.9
Gen. 5	15.1	3.14	20.7	13.7	3.35	24.4
SMALLS, Excluding dwarfs						
Gen. 1	17.8	2.30	12.9	18.2	2.22	12.1
Gen. 2	17.4	2.28	13.1	16.4	2.39	14.5
Gen. 3	16.1	2.01	12.4	15.2	2.18	14.3
Gen. 4	14.3	1.96	13.7	14.7	2.26	15.3
Gen. 5	15.7	2.09	13.3	15.0	2.28	15.2
MEDIUM						
Gen. 1	23.7	2.24	9.4	22.9	2.23	9.7
Gen. 2	24.0	1.99	8.2	24.5	2.15	8.7
Gen. 3	21.6	1.67	7.7	21.8	1.73	7.9
Gen. 4	22.8	2.15	9.4	23.1	2.34	10.1
Gen. 5	23.4	1.78	7.6	24.0	2.31	9.6
LARGE						
Gen. 1	27.4	2.74	10.0	27.4	2.62	9.5
Gen. 2	31.2	2.47	7.9	29.9	2.75	9.1
Gen. 3	28.6	3.59	12.5	26.6	2.58	9.6
Gen. 4	31.0	3.28	10.5	30.0	3.20	10.6
Gen. 5	31.8	2.88	9.0	33.9	3.51	10.3

*Includes individual and litter variance but not that due to breeding type, sex, site group, or inbred line.

were dwarfs in generations three to five inclusive. If mating had been at random within each of these groups and if the condition was due to a single recessive gene, this would correspond to a gene frequency of over 39 percent.

The original stocks were thought to be free of deleterious recessive genes. Later, however, Butler[1] stated that he obtained stocks from Dr. MacArthur in 1948 and found that a dwarf gene was present in the small stock. King[5] also observed dwarfs in the progeny of stock obtained at about this time and called the condition "pygmy."

It seems probable that our original stocks carried the same gene observed by Butler and King in low frequency and that it has some effect in the heterozygous condition. Selection for small size then increased its frequency.

Summary

Different groups from a mouse population were selected for small, medium, and large 60-day weight, respectively, for five generations. Each size group included four inbred lines, each ideally maintained as a one-sire line of 10 breeding females, and an outbred population descending from the same foundation animals as the inbreds. Data on 8641 animals are included in the analysis.

Selection for large and small 60-day weight was effective in both inbred and outbred populations. Heritability of 60-day weight was slightly lower in inbreds than in outbreds, but there was no evidence of inbreeding depressing heritability more than would be expected from the fixation of genes occurring with inbreeding.

Selection did not affect variability appreciably in any size group, except as the occurrence of dwarfs in the small group tended to increase it.

Litter size tended to decrease in all size groups as the experiment progressed. The reason for this is unknown.

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A USEFUL CHROMOSOME MODEL FOR LABORATORY DEMONSTRATIONS

Thomas M. Little*

In the course of teaching a laboratory course in genetics, the writer has devised a very simple chromosome model which has proved useful in demonstrating linkage and crossing-over.

The only materials needed for this model are three or four silver dollars and a piece of rubber tubing three or four feet in length. Slits are cut in the rubber tubing about 1⅜ inches in length and the silver dollars are inserted in the slits and held in place with a short piece of transparent tape on each side. Care must be exercised in making all of the slits on the same plane, in making them parallel to the walls of the tube and in having them perfectly centered. A fairly flexible type of rubber tubing should be used, and the most satisfactory size is one with a bore diameter of ¼ inch and a wall thickness of 1/16 inch. The minimum distance between the coins depends on the flexibility of the tubing. This distance can be found by making one slit about an inch from the end of the tube and inserting a coin, then determining how close to this coin a twist of 180 degrees can be applied to the tubing without producing enough torsion to turn the coin over. In order to illustrate the principle that the frequency of crossing-

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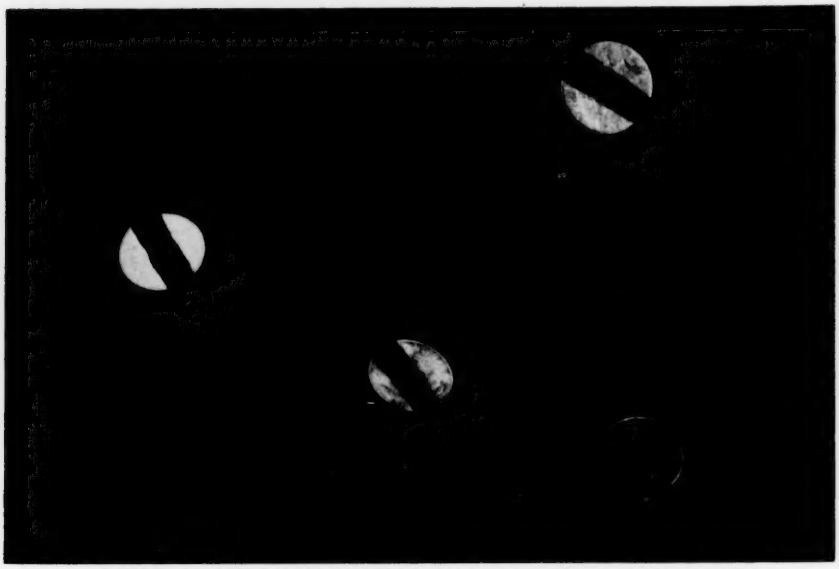

A MODEL FOR DEMONSTRATING LINKAGE AND CROSSING-OVER

Figure 8

Rubber tubing with silver dollars inserted, represents a pair of chromosomes at meiosis, with each coin representing a heterozygous pair of alleles. Heads designate the dominant allele and tails the recessive, or vice versa. The model is "tossed" just like a single coin.

over is proportional to the distance between the genes, it is well to vary the spacing between the coins on the rubber tubing.

The tubing with the coins inserted can then be considered to be a model of a pair of chromosomes at meiosis, with each coin representing a heterozygous pair of alleles, one side (for example, heads) being designated as the dominant allele and the other side as the recessive. The normal tubing with no twists represents a pair of chromosomes with no chiasmata, and the coin faces exposed on each side then represent the parental combinations of genes. A twist of the tubing between any two consecutive coins represents a cross-over in that region of the "chromosome."

This model may be tossed just as one tosses individual coins, being careful to make the tosses in a random manner rather than using any systematic procedure. It is well to use a rug or mat, otherwise the coins will cut through the tubing if the model is tossed onto a hard surface. After each toss the coins are read in a fixed order and recorded, and when sufficient tosses have been recorded the data can be treated like any set of linkage data. For the sake of simplicity, we generally have all of the coins oriented in one direction, representing the parental combinations of genes *ABCD* and *abcd*. However, it is possible to introduce the notion of coupling and repulsion by reversing one or more of the coins. Another effective variation of the procedure is to designate alternating tosses as male and female gametes and to record the zygotes resulting from each pair of tosses. These data can then be used as F_2 linkage data.

One of the most impressive demonstrations we have conducted with these models was to have two laboratory sections exchange data and calculate the order of the "genes" on the "chromosome" and the parental gene combinations. The close correspondence between the "genetic map" based on the data and the "cytological map" based on the actual model is very striking.

Precautions should be taken to avoid one source of bias that sometimes accompanies use of these models. If the terminal coins are not perfectly centered they may land more or less frequently than expected by chance. This bias can be overcome simply by agreeing at the start of an experiment to designate the heads of the coins as dominants during the second half.

Table I shows some actual data secured during one laboratory period using a model with four coins all oriented in the same direction. The model was tossed 512 times and the gametes recorded, and also the phenotypes resulting from each pair of tosses gave an F_2 population of 256 individuals. The coins in this model

were placed fairly close together so that over the whole length of the model there were no twists or cross-overs in 57.8 percent of the tosses. It will be noted that the cross-over values from *A* to *C*, *B* to *D* and *A* to *D* are very close to the values one would expect when taking into account the expected frequencies of double cross-overs.

Table II shows the results obtained with a model in which the coins were spaced somewhat farther apart than in the model mentioned above. Table III gives data from a model in which the coins were still more distantly spaced. As a matter of fact, only the data for genes *C* and *D* show a significant deviation from independent assortment. Spacings as wide as this do not afford a good demonstration of linkage, but this type of model does bring out the principle that the upper limit of crossing-over is 50 percent regardless of the length of the chromosome segment involved.

Table IV shows the data from a simpler type of model than the ones used above, since only three coins were used instead of four. For general demonstrations in elementary genetics, this model is probably preferable.

TABLE I. Data from chromosome model with four "genes" spaced fairly close together

	Gametes		Phenotypes	
	Observed	Expected*	Observed	Expected*
ABCD	153	148.2	144	140.7
ABCd	29	32.0	14	16.2
ABcD	11	8.8	7	7.3
ABcd	39	40.5	17	15.2
AbCD	3	4.7	2	4.2
AbCd	0	1.0	1	0.6
AbcD	4	3.7	3	2.5
Abcd	20	17.1	6	5.2
aBCD	17	17.1	10	9.3
aBCd	1	3.7	2	1.3
aBcD	2	1.0	4	0.7
aBcd	6	4.7	3	1.4
abCD	40	40.5	12	17.1
abCd	9	8.8	3	2.6
abcD	35	32.0	12	10.3
abcd	143	148.2	16	21.4
TOTAL	512		256	

*Expectancies based on cross-over values between adjacent coins.

Cross-over values: A-B 10.35%; B-C 21.48%; C-D 17.77%; AC 27.54%; BD 30.66%; A-D 35.54%.

TABLE II. Data from model with four "genes" spaced a medium distance apart

	Gametes		Phenotypes	
	Observed	Expected	Observed	Expected
ABCD	86	78.2	110	105.7
ABCd	42	46.7	27	26.4
ABcD	32	28.8	20	20.5
ABcd	44	48.3	12	13.2
AbCD	12	11.5	11	10.6
AbCd	7	6.8	1	2.9
AbcD	14	11.1	8	5.0
Abcd	13	18.6	1	3.2
aBCD	20	18.6	15	13.4
aBCd	10	11.1	5	3.4
aBcD	6	6.8	3	3.0
aBcd	14	11.5	2	1.9
abCD	37	48.3	14	19.9
abCd	39	28.8	8	5.3
abcD	37	46.7	10	9.5
abcd	87	78.2	3	6.1
Total	500		250	

Cross-over values: A-B 19.2%; B-C 38.2%; C-D 37.4%; A-C 41.8%; B-D 42.8%; A-D 41.2%.

TABLE III. Data from model with four "genes" spaced long distances apart

	Gametes		Phenotypes of Zygotes	
	Observed	Expected	Observed	Expected
ABCD	36	36.6	83	70.3
ABCd	21	19.0	13	16.3
ABcD	15	16.6	19	15.3
ABcd	33	31.8	20	11.7
AbCD	35	29.4	13	21.0
AbCd	11	15.3	1	5.0
AbcD	27	17.6	7	5.9
Abcd	35	33.8	4	4.5
aBCD	26	33.8	17	22.5
aBCd	16	17.6	4	5.2
aBcD	13	15.3	7	4.9
aBcd	29	29.4	7	3.8
abCD	29	31.8	2	7.8
abCd	21	16.6	0	1.9
abcD	13	19.0	2	2.2
abcd	40	36.6	1	1.7
TOTAL	400		200	

Cross-over values: A-B 48.0%; B-C 46.5%; C-D 34.25%; A-C 50.5%; B-D 50.75%; A-D 45.25%.

TABLE IV. Data from model with three "genes"

	Gametes		Phenotypes of Zygotes	
	Observed	Expected	Observed	Expected
ABC	87	78	98	95.6
ABc	42	42	26	22.4
AbC	24	28	14	18.5
Abc	54	52	10	13.5
aBC	45	52	23	25.6
aBc	37	28	6	6.4
abC	37	42	14	10.4
abc	74	78	9	7.6
TOTAL	400		200	

Cross-over values: A-B 40.0%; B-C 35.0%; A-C 44.5%.

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THE INHERITANCE OF MALOCCLUSION DUE TO MANDIBULAR PROGNATHISM

KARL A. STILES AND JOHN E. LUKE*

PROTRUDING JAW

Figure 9

Photograph and radiograph of individual III-3 (Figure 10) shows characteristic protruding lower jaw although prognathism is not pronounced. The arrow in the radiograph points to the mandibular angle formed at the junction of the body and ramus of the lower jaw thus causing the abnormal protrusion.

OUR knowledge of the relation of genetics to malocclusion leaves much to be desired; however, some progress is being made.

In 1923 Dr. M. F. Guyer[2] gave a paper at a meeting of the Dewey School of Orthodontia, in which he said, "I find myself coming to you almost empty-handed as regards any accurate knowledge regarding the inheritance of dental and facial conditions." A review of the literature, since the Guyer paper was given, shows that many contributions have been made in this general field, but not much with reference to the problems of malocclusion. For the most part, contributions in dental genetics have concerned special traits pertaining to the teeth themselves, such as absence of certain teeth or deficient roots of teeth, hypoplasia of enamel, microdontia, supernumerary teeth, susceptibility to dental caries and so on. In the literature, there are well over a hundred references to the inheritance of such dental traits. So we have not moved very far from the empty-handed stage, of which Guyer spoke, insofar as genetic work in orthodontics is concerned. A rather thorough search of the literature on dento-facial anomalies, made in the course of this study, included besides the various jour-

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nals of genetics, the *Index Catalogue Surgeon General's Library* 1880 to date, the *Index Medicus* 1916 to 1950, and the *Current List of Medical Literature*, July, 1949 to April, 1952. Yet, very little reliable data on pedigree studies of malocclusion due to mandibular prognathism were found. However, statistical investigations such as Iwagaki's[3] leave no doubt as to the existence of a hereditary component in this type of prognathism, but his extensive studies throw no light on the exact mode of inheritance. Twin research on the relation of heredity to mandibular prognathism by Ritter[7] and others shows that concordance in monozygotic twins is much more frequent than in dizygotic twins.

Faulty closure of the upper and lower jaws results in some form of malocclusion. During the growing period of an individual, the component parts of the whole oral mechanism are highly coordinated to produce an enlarging yet relatively stable pattern of development. Any deviation from the normal developmental pattern, however, may result in one of the different types of malocclusion.

Numerous factors can bring about such an abnormal development of the oral mechanism, such as endocrine disturbance, anodontia, maldevelopment of the mandible or maxillary arch, injury to the oral mechanism, or early loss of teeth. Thompson[12] points out that one of the various component parts of the face and cranial base may be deficient, or there may be a combination of deficient parts which cause the malocclusion. How malocclusion results from each of these factors is beyond the scope of this paper. That heredity plays an important role in some types of malocclusion is shown from investigations by Rubbrecht,[10] who collected several pedigrees of malocclusion; Macklin and Monroe,[5] who made a detailed study of the occurrence of malocclusion in a pair of identical twins; and Moore and Hughes,[6] who studied the inheritance of various types of malocclusion in over a hundred families. These investigators concluded that although environmental factors might influence the occlusion of an individual, the evidence is strong that it is usually due primarily to hereditary factors. Although there are many conditions which can bring about different forms of malocclusion, this study was limited to the relation of genetics to the etiology of mandibular prognathism.

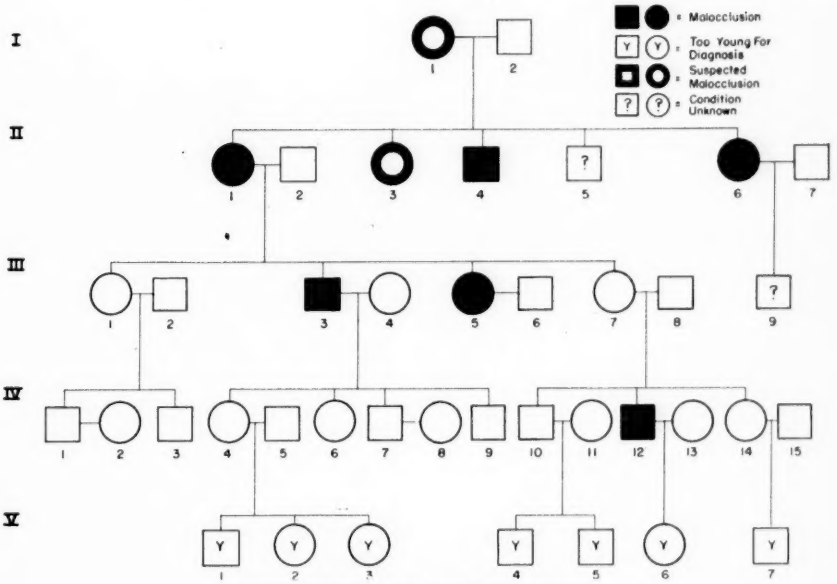
Mandibular prognathism is a dentofacial anomaly characterized by protrusion of the mandible (Figure 9). This condition sometimes gives the afflicted individual a "bull dog" appearance, because the lower incisors often overlap the upper incisors. A person with a greatly exaggerated prognathic jaw is sometimes called lantern-jawed. This protruding lower jaw is not caused by a dental or alveolar protrusion, but rather by a forward positioning of the mandible itself.[1] Forward positioning of the mandible may be caused by one of two distinctly different developmental processes. One form, which is not mandibular prognathism in the true sense of the word, is caused by a foreshortening of the maxillary arch, thereby causing the normal mandible to project beyond the micro-developed upper jaw. The other type, which is considered by most workers as true mandibular prognathism, is brought about by an increased obtuseness in the angle* of the mandible (Figure 9), producing a protrusion of the mandible beyond the normal maxillary arch.[6] These two distinct types of mandibular protrusion should not be confused, for each is caused by a different developmental process and may be inherited independently of the other.

Notes on the Pedigree

This family of five generations (Figure 10) has been carefully studied in regard to the inheritance of malocclusion due to mandibular prognathism. In view of the fact that conclusions can only be as valid as the data on which they are based, it seems worthwhile to indicate some of the problems encountered in collecting the data.

I-1 has been deceased for some time, but information obtained from relatives and pic-

*Mandibular angle as used in this paper refers only to the angle between the body and the ramus of the lower jaw.



INHERITANCE OF MALOCCLUSION
Figure 10

The malocclusion observed in this family appears to be due to a forcing of the lower jaw forward by reason of an increased obtuseness of the mandibular angle. The mandibular angle appears to be determined primarily by the genotype of the individual.

tures all gave evidence that she had a prognathic mandible; and the authors are convinced that if dental examination had been possible, this finding would have been confirmed. However, because she was unavailable for dental examination, she is classified in the pedigree chart (Figure 10) as suspected of having a prognathic lower jaw. I-2 was diagnosed from good photographs as nonprognathic. II-1 showed a definite undershot jaw; this diagnosis was confirmed when she was examined by a dentist. II-2 was diagnosed from photographs as nonprognathic. II-3 lived in Europe all of her life and only a few relatives remember her. Photographs, however, did give evidence of a prognathic mandible; but because an unquestionable diagnosis could not be made, she is designated in the pedigree chart as suspected of having malocclusion. II-4 diagnosis was not professionally determined since he too lived all his life in Europe; but because he was observed by so many relatives who reported a pronounced prognathic mandible, there seemed no doubt about his condition. II-5, like his brother and sister, spent his life in Europe. He is reported by some relatives as not possessing a prognathic mandible, but there is some question about the reliability of these reports; therefore, his condition is classified as unknown. II-6 is reported by relatives as troubled by a protruding mandible, and photographs leave no doubt of the correctness of these reports. II-7 was diagnosed from the examination of good photographs as being free from mandibular prognathism.

The diagnoses of all individuals in generations III through V, except III-9, have been made by a competent dentist. Furthermore, they have also been examined by one of the authors.

The diagnosis of mandibular prognathism from photographs suffers several limitations. As might be expected, there was some variation in the development of the prognathic jaw in this family, from one individual to another; for example, it is more pronounced in IV-12 than in III-3. Then there must also be recognized the possibility that a photograph of an adult may be that of a person who in adolescence had a prognathic condition corrected by orthodontics. However, it seems very improbable that individuals in either generation I or II ever had the benefit of orthodontics.

Discussion

The pedigree chart shows that mandibular prognathism occurs in at least three, and probably four of the generations. Because the condition may not come to expression until adolescence, it is impossible to determine at this time whether individuals of the fifth generation

are going to exhibit the trait for they are all under eight years of age.

According to Korkhaus,[4] mandibular prognathism affects one-half of one percent of the population that has reached the age of 14 years. If this is true, then the probability is not very great that persons marrying prognathic individuals would be carriers of the gene. If this assumption is correct, then I-2 would probably not be a carrier of the gene for prognathic mandible and it would follow that the prognathic jaw of II-1, II-4, and II-6 is probably due to the inheritance of a dominant gene from I-1.

Furthermore, the same reasoning would hold for II-2. It would seem improbable that he is a carrier, and the same is true for III-4 and III-8. Even if we admit some probability of one or two of these individuals being carriers, nevertheless to assume that all of them are carriers appears to be a rather improbable hypothesis. If the prognathic jaw is caused by a dominant gene, there is evidence that it does not always come to expression, for III-7 is probably a carrier but does not show the trait.

Thus it appears that this pedigree favors the conclusion that the gene in question is dominant with incomplete penetrance. Some other studies of mandibular prognathism, including those by Rubbrecht[8, 9] of the Hapsburg Dynasty of Europe, support this conclusion. The literature speaks of the Hapsburg lip, but what is more striking to a student of dentofacial anomalies is the prognathic jaw of the Hapsburgs. Also, another family under investigation by the authors at this writing seems to provide additional evidence of dominant inheritance of the prognathic mandible. Insofar as we could find from the literature, there is no good evidence of this condition resulting from recessiveness. However, on the basis of our present pedigree data, we cannot exclude the possibility of recessive inheritance. We plan to make this a longitudinal study so this paper must be considered only as a progress report. Ten years from now the fifth generation in this pedigree may yield some critical information. The practical importance of knowledge regarding the inheritance of the prognathic lower jaw is quite obvious. Hereditary influences may operate to negate beneficial changes produced by the usual orthodontic methods. However, malocclusion due to this mandibular defect can be corrected in the majority of cases by means of appliances, surgery, or both. In early adolescence braces may prove entirely adequate for correcting mandibular prognathism, but in late adolescence or adulthood the possibilities for correction may be poor. In fact, in adulthood the only satisfactory method of treatment is the surgical removal of sections of the mandible. In families where this dentofacial defect is known to be a dominant trait, it should be anticipated in some of the children and diagnosed at the earliest possible age when orthodontic treatment is most effective. Furthermore, if a dental defect is known to exist in one or both of the parents, it may help to determine the probable course of the same condition in the child. Snodgrasse[11] draws the conclusion from his investigations "that a consideration of heredity and growth variables will assist the orthodontist in the expeditious and successful treatment of patients." Thus the evidence is growing that genetics can aid the dentist, oral surgeon, and orthodontist by giving them a tool which enables them to make earlier diagnosis and better prognosis, and thereby provide more satisfactory treatment.

Summary

1. Mandibular prognathism is the cause of one type of malocclusion. There is a protrusion of the mandible and the lower incisors may occupy a position anterior to the upper incisors. This condition may be caused by an underdeveloped maxilla or an increased obtuseness in the mandibular angle.

2. This pedigree study favors the conclusion that mandibular prognathism, caused by increased obtuseness of the mandibular angle, is inherited by means of a dominant gene with an unknown degree of reduced penetrance.

3. Some other studies of mandibular prognathism including the Hapsburg Dynasty of Europe appear to support the conclusion that the prognathic mandible is due to a dominant gene with incomplete penetrance.

4. Mandibular prognathism can be corrected in the majority of cases by means of surgery, appliances, or both. The earlier the diagnosis, the easier treatment becomes; and a knowledge of the genetics of the condition may help the orthodontist to anticipate some of the difficulties he may encounter in the treatment of a patient, and to predict to some extent the degree of success of the treatment which he applies.

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Backcrossing for Plant Breeders

THE title of this small book* of 136 pages would lead one to believe that it is a general, theoretical discussion of the backcross method. This assumption is misleading; the subtitle makes clear that the discussion is confined to the theory and practice of the backcross method as used in the improvement of certain non-cereal crops. Four crops—cotton, tobacco, tomato and potato are treated; the length of treatment for each crop being roughly proportional to the amount of experimental work in which the backcross method has been used, (55 pages for cotton, 25 for tobacco, 23 for tomato, and 17 for potato).

After a brief chapter of four pages on the theory of backcrossing, the text is concerned mainly with a non-critical, detailed account of the transfer of a gene or group of genes from the donor parent to the recurrent parent. These genes are for the most part those that control such economic characters as disease and insect resistance, fiber length in cotton, nicotine content in tobacco, frost resistance in potato, etc.

In many instances the author had to compile her story from the annual reports of a number of agricultural experiment stations. This is a difficult task, requiring a great deal of critical discrimination. Most annual reports lie comfortably buried on the library shelves of experiment stations. This is as it should be, as they are likely to be more enthusiastic than accurate. Forced to rely on uneven material of this sort, it is not surprising that some inaccurate statements have made their way into the book. For example, the author states that the strain of *Lycopersicon pimpinellifolium* designated P.I. 79532 from which the *Fusarium* wilt variety Pan America was developed is different from Missouri Agricultural Experiment Station, Accession 160 (incorrectly given as 106, pg. 89) strain of *L. pimpinellifolium*. Dr. G. W. Bohn, a co-discoverer of the dominant gene for *Fusarium* wilt resistance at Missouri tells me that P.I. 79532 and Accession 160 were the same, and presumably carried identical genes for *Fusarium* resistance.

For some unknown reason, a paper by Clayton and Graham on breeding for resistance to root knot and nematode root rot in tobacco is reviewed in the chapter on tobacco (pg. 78, IX) and repeated with minor variations in the chapter on tomato (pg. 103, XIV).

The bibliographies are conveniently arranged at the end of each chapter. There are 177 references on cotton; 74 on tobacco; 117 on tomato and 52 on potato. The index is useful and accurate. Plant breeders and geneticists interested in the four crops under review will find this book helpful, if for nothing more than the citations of the literature.

This reviewer feels that the author has missed an exceptional opportunity to evaluate the theoretical basis of the backcross method. If theory had been combined with a critical appraisal of the practical results that are so well described, it would have added much to the book's significance.

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**Back Crossing*. The Theory and Practice of the Backcross method in the breeding of some non-cereal crops. Mary Thomas, i-vi, 1-36. Technical Communication 16; Commonwealth Bureau of Plant Breeding and Genetics. Cambridge, 1952.

GENETICS OF SEVERAL EYE COLOR MUTANTS IN THE HONEY BEE

Apis mellifera L.

H. H. Laidlaw, M. M. Green and W. E. Kerr*

MALE honey bees with "white" eyes or eyes of other colors have, for many years, been reported to occur in occasional colonies. The study of these aberrant types has been difficult because the queen honey bee mates in flight and will not mate in enclosures. Consequently almost no genetic studies of the honey bee were made until comparatively recently when a technique of artificial insemination became available to the geneticist.

The honey bee females are diploid and the males haploid. The males develop from unfertilized eggs which have undergone typical maturation with its attendant segregation. The drones are thus genetically gametes of their mother. Characters which are recognizable in drones can be used to establish segregation ratios from queens and thereby eliminate the necessity for backcrossing for this purpose.

A recent review of the more pertinent literature pertaining to the genetics of the honey bee[5] indicates a striking paucity of genetic information. In their study of the origin of gyndromorphs in the honey bee Rothenbuhler *et al.*[3] have used eye color mutants designated as "ivory", "cream", and "chartreuse", which were discussed briefly in another paper by the same authors.[4]

Source and Description of the Mutants

The following listing of the eye color mutants which are to be discussed includes the name, proposed gene symbol and a description of the eye color. Eye color classifications according to Munsell colors are included. A fruit grading lamp standardized at 5500° Kelvin was used in making these classifications. The frequency of occurrence of each mutant among the drone progeny of the original queen in which the mutation was discovered, except as noted, is also recorded.

Chartruese-1 (ch^1). The eye color in newly emerged individuals is chartreuse and ranges from 7.5 Y 8/6 to 10.0 YR 5/6. The ventral eye tips of the darker chartreuse individuals are brownish, and tips of the lighter chartreuse individuals are brownish or sometimes brownish-green. The eye color darkens with age to reddish-brown by the encroachment of pigment from the eye margins, finally reaching dark red-brown in queens. The original mutants occurred among drone progeny of a single black-eyed queen in the following frequency: black 528; chartreuse 364; brown 407. It may be presumed from this that the queen was heterozygous for the ch^1 mutant. The nature of the concomittant brown phenotype reported will be discussed later.

Chartreuse-2 (ch^2). The eye color in newly emerged individuals is chartreuse and ranges from 7.5 Y 8/6 to 5.0 Y 7/6, and rarely to 2.5 Y 6/4. Eye tips of most individuals are greenish, some of the darker are brownish-green. On aging, the eyes darken to reddish-brown, and then to dark reddish-brown in queens. Original mutants appeared among the drone progeny of a single black-eyed queen in the frequency: black 617; chartreuse 590. It is thus probable that the queen was heterozygous for the mutant.

Red (ch^r). The eye color in newly emerged individuals is red with a purplish tinge and ranges from 7.5 R 2/4 to 2.5 R 2/6. The eye tips are slightly darker. On aging the eyes darken to very dark reddish-brown. The original

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mutant drones were brought to the laboratory by a beekeeper and the frequency in which they occurred is unknown although the parental queen is known to have been black-eyed.

Brick (*bk*). Eye color in newly emerged individuals is brick red with an orange tinge and ranges from 10.0 R 4/4 to 7.5 R 3/6. The eye tips are slightly darker. On aging the eyes darken to reddish-brown. The original mutants appeared among the drone progeny of a single black-eyed queen in the frequency: black 2878; brick 15. The mutant apparently arose spontaneously in one ovariole of this queen or possibly in a stem cell prior to ovariole formation.

Ivory (*i*). The eye color is ivory white in newly emerged individuals and darkens only slightly with age. Red never appears in the eye at any time which indicates a complete inhibition of pigment formation. This mutation was obtained from Dr. Otto Mackensen, Division of Bee Culture and Biological Control, United States Department of Agriculture.

A recessive body color mutant, cordovan, (*cd*), which causes cuticular areas that are normally black to be brown was also obtained from Dr. Mackensen.

Methods of Securing and Counting Individuals

Virgin queens were reared by the usual queen rearing methods[2] and the cells were placed in nuclei on the tenth day after grafting. In the case of queens to be artificially inseminated, queen excluders were placed over the entrances of the nuclei to prevent the queens from flying, and on the second day after emergence the wings on one side of the queens were clipped to further insure against flight. Whenever female progeny were required the queens were artificially inseminated.[1] Usually semen from several "brother" drones was injected into each queen.

Drones for mating were obtained in most cases as follows: the drone mothers were provided with drone comb. After three to six days the drone combs which then contained eggs, or eggs and larvae, were transferred to a feeding colony. Two days prior to drone emergence the combs of drone brood were caged in special cages and either left in strong colonies or were put into an incubator. As soon as the drones emerged in sufficient numbers they were transferred to maturing cages which were then put into special feeding colonies. The drones were ready for use in artificial insemination eight to ten days after emergence. Many of the heterozygous queens were marked by numbered discs glued to the mesonotum of the thorax and were permitted to mate naturally.

The individuals to be classified, both male and female, were reared as above and were counted on the day or the day following emergence. Where it was necessary to distinguish between similar colors, the classifications were made with the aid of the fruit grading lamp as noted previously.

Tests for dominance. To test the dominance relationships between the eye color mutants and normal black eye color, mutant drones were crossed by artificial insemination to black-eyed queens derived from lines which previously had produced only black-eyed progenies and which may be assumed to be homozygous black-eyed. The phenotypes of the worker progenies of these crosses are listed in Table I. In all cases the F_1 workers were black-eyed in phenotype indicating that each of the eye color mutants tested is recessive to black.

Tests for allelism. In a similar fashion the allelic relationships of the eye color mutants were tested. Crosses between queens homozygous for a particular mutant and drones of a different mutant were made by artificial insemination, and the phenotypes of the worker progenies were determined. The results of these crosses are also included in Table I. It may be noted that workers of the genotypes ch^1/ch^r and ch^2/ch^r were intermediate in eye color, and those of the genotype ch^1/ch^2 were chartreuse. The phenotypes of ch^1 and ch^2 are difficult to separate but data to be reported subsequently will demonstrate that ch^1 and ch^2 do not represent identical mutants. These results suggest that ch^1, ch^2, and ch^r constitute an allelic series. In the other tests among the

TABLE I. F_1 worker phenotypes from cross of queens and drones as indicated

Phenotype of queen parent	Phenotype of drone parent					
	black	chartreuse-1	chartreuse-2	red	brick	ivory
black	black	black	black	black	black	black
chartreuse-1		chartreuse	chartreuse	intermediate	black	black
chartreuse-2			chartreuse	intermediate	black	black
red				red	black	black
brick					brick	black
ivory						ivory

eye color mutants, the F_1 workers were black-eyed, and it can be assumed that the mutants *bk* and *i* involve loci non-allelic to *ch* and non-allelic to each other.

Segregation and Linkage Tests of Eye Color Mutants

Segregrations observed among the drone progenies of queens heterozygous for the eye color mutants confirm the hypothesis that the eye color phenotypes tested are conditioned by simple, mendelizing mutants. The heterozygous queens used in these tests were daughters of the queens listed in Table I and were, therefore, F_1 queens. These queens were either artificially inseminated or were marked and permitted to mate naturally. It should be emphasized that the mating of the F_1 queens had no influence whatever on the drone progenies since drones develop from unfertilized eggs. The male progenies of the F_1 queens represent the result of gametic segregations and are not strictly speaking F_2 progenies.

Drones reared from the F_1 queens were classified and enumerated according to phenotype. The segregation of each mutant and its dominant black allele are listed in Table II. Since drones are derived from queen gametes, a typical monohybrid segregation ratio is 1:1. The segregation frequencies obtained, meet this expected ratio in all cases except in tests (1) and (6) involving chartreuse-1 and brown respectively. Queens in each of these tests produced three phenotypic classes of drones instead of the expected two. It may be recalled that the *ch¹* mutant was originally detected among the drone progeny of a black-eyed queen which produced three phenotypically separable classes of drones, viz., black, chartreuse, and brown. In order to explain the above exception to the expected 1:1 ratio and the observed frequencies of occurrence of the different phenotypes, it is necessary to assume that the brown phenotype results from the interaction of *ch¹* and a non-allelic, independent, recessive modifier gene which can be designated *m*. With this assumption the brown-eyed drone genotype may be designated *ch¹; m* and the chartreuse-1 drone genotype *ch¹; m+*.

On the basis of the above assumptions the genotype of the F_1 queen of test (1) was *ch¹/ch+; m/m+*, and it would be expected that among her drone progeny ¼ would be *ch+; m* and ¼ *ch+; m+* which together would be ½ black-eyed. It would also be expected that ¼ would be *ch¹; m+* or chartreuse, and ¼ *ch¹; m* or brown. The results as listed for test (1) in Table II are compatible with this interpretation.

The drone frequencies obtained from heterozygous queens in tests (6) and (7) of Table II are similarly compatible with the above interpretation. The heterozygous queen of test (6) produced three phenotypic kinds of drones, viz., black, brown, and chartreuse in the ratio 2:1:1. The heterozygous queen of test (7) which was presumably identical, in respect to the eye color genes, to the queen of test (6) produced only two types of drones: black and brown in the ratio 1:1. These two F_1 queens must have differed in regard to their *m* genes. The queen of test (6) must have been heterozygous at both the *ch¹* and *m* loci and therefore had the genotype *ch¹/ch+; m+/m*. Accordingly her drone progeny consisted of black, chartreuse and brown eyed individuals in the frequency of 2:1:1. The queen of test (7) must have been heterozygous for the *ch¹* mutant but homozygous for the *m* gene and therefore she had the genotype *ch¹/ch+; m/m*. Consequently her drone progeny were

TABLE II. Segregation of drone progeny from heterozygous queens

Source of queens (black ♀ × mutant ♂ as listed)	Phenotypes and frequencies of drone progenies*						Total drones
	black	chartreuse	red	brick	ivory	brown	
(1) chartreuse-1 ♂	1242 (1223)	601 (611.5)				603 (611.5)	2446
(2) chartreuse-2 ♂	80 (80.5)	81 (80.5)					161
(3) red ♂	71 (80.5)		90 (80.5)				161
(4) brick ♂	301 (316.1)			322 (316.1)			623
(5) ivory ♂	1049 (1056)				1063 (1056)		2112
(6) brown ♂	498 (500)	272 (250)				230 (250)	1000
(7) brown ♂	140 (141)					142 (141)	282

*Numbers in parentheses represent expected frequencies.
χ^2 tests indicate observed and expected frequencies not significantly different.

expected to be black and brown in a ratio of 1:1 as was observed.

The available data indicate that the *m* gene fails to affect the *ch^r* phenotype and affects *ch^2* only slightly. This is shown in tests (12) and (16) of Table III. The heterozygous queen of test (12) produced drone progeny consisting of chartreuse and brown in a ratio of 3:1. Assuming the *m* gene affects *ch^1* and *ch^2* in a similar fashion, it would be expected that the drone progeny of this queen would consist of equal frequencies of brown and chartreuse. However, if *m* does not affect *ch^2*, a ratio of 3 chartreuse : 1 brown would result, and this was actually the case. It may be added that in classifying her drones approximately ¼ were assigned as *ch^2* and ½ *ch^1* in phenotype. Since the expected ¼ brown drones were observed, it can be presumed that the excess of drones of the *ch^1* phenotype is the result of a slight modifying action of *m* on *ch^2* resulting in a *ch^1* phenotype. Thus the *ch^1* class of drones included the expected ¼ *ch^1*; *m+* and probably the ¼ *ch^2*; *m* genotypes. This differential action of *ch^1* and *ch^2* in the presence of *m* indicates that while *ch^1* and *ch^2* produce essentially identical phenotypes they are in fact distinct alleles and do not represent separate mutations to the same allele.

That the modifier *m* does not affect the *ch^r* gene is similarly concluded from test (16) (Table III). The drone progeny of this heterozygous queen approximated a ratio of 1 chartreuse : 2 red : 1 brown. This is expected if the *m* gene fails to influence *ch^r*, whereas if *m* did modify *ch^r* a fourth phenotype would be expected.

It may be added that the segregation results involving *ch^1*, *ch^2*, and *ch^r* confirm the proposition made earlier, based on phenotypic interaction, that these three mutants constitute an allelic series.

Tests made to detect linkage between the various eye color mutants were in all cases negative (Table III). It may be noted that in tests involving *ch^2* and *i*, and *ch^r* and *i* (tests 11 and 15 respectively) essentially identical results were obtained. In the case of *ch^2* and *i* the phenotypic segregation among the drones approximated a ratio of 1 black : 1 chartreuse : 2 ivory, and similarly with *ch^r* and *i* the phenotypic ratio approximated the ratio 1 black : 1 red : 2 ivory. These results indicate that *ch^2* and *ch^r* genes assort independently of the *i* gene and that *i* is epistatic to *ch^2* and *ch^r*.

Tests involving *ch^2* and *bk*, and *ch^r* and *bk* (tests 10 and 14 respectively) indicate that the *ch* and *bk* loci assort independently. Although a chi-square test indicates that the observed results of test (10) deviate significantly from expectation at the 2 percent probability level $\chi^2 = 11.1$, $p = 0.01 - 0.02$) this is not considered to indicate linkage. In a comparable test using the *ch^r* allele and *bk* (test 14) results compatible with independent assortment of the two loci were obtained ($\chi^2 = 5.04$, $p = 0.1 - 0.2$). The buff phenotype segregating in test (10) probably represents drones of the genotype *ch^2*; *bk*, and the pink phenotype of test (14), probably represents drones of the genotype *ch^r*; *bk*.

In order to test the linkage relationships between *i* and *ch* loci and the gene for cordovan body color *cd*, F_1 heterozygous queens

TABLE III. Tests for linkage between the eye color mutants

Genotype of queen	Phenotypes and frequencies of drone progenies* black	chartreuse	red	brick	ivory	brown	pink	buff	Total drones
(8) *ch^1/ch^2*		1737 (1737)							1737
(9) *ch^2/ch^r*		439 (439)	439 (439)						878
(10) *ch^2/ch+; bk/bk+*	477 (418.75)	401 (418.75)		400 (418.75)				397 (418.75)	1675
(11) *ch^2/ch+; i/i+*	343 (358)	378 (358)			711 (716)				1432
(12) *ch^2/ch^1; m/m+*	1†	358 (360)				122 (120)			480
(13) *ch^r/ch^1*	2†	1005 (1006.5)	1008 (1006.5)						2013
(14) *ch^r/ch+; bk/bk+*	294 (262.5)		252 (262.5)	256 (262.5)			248 (262.5)		1050
(15) *ch^r/ch+; i/i+*	167 (152.25)		128 (152.25)		314 (304.5)				609
(16) *ch^r/ch^1; m/m+*		394 (381.25)	726 (762.5)			405 (381.25)			1525

*Numbers in parentheses represent expected frequencies.
†Probably from laying workers; not included in totals.
χ^2 tests indicate observed and expected frequencies not significantly different except (10). See text for discussion.

were produced from the cross female normal body color, chartreuse-2 × male cordovan, ivory. The male progeny reared from the F_1 queen were classified and the results listed in Table IV. These results do not deviate significantly from expectation based on the independence of the three loci.

While the data obtained from these tests fail to demonstrate linkage between any of the eye color mutants (linkage tests of *i* and *bk* not completed), they do not exclude the possibility of linkage. A situation of very loose linkage might conceivably exist among the mutants reported here and escape detection because of the lack of appropriate genetic markers.

TABLE IV. Test for linkage between *ch*^{1}, *i* and *cd* mutants (Drone progeny from F_1 queen from cross: ♀ normal body; chartreuse-2 × ♂ cordovan, ivory.)

Phenotypes and frequencies of drone progeny*						
Normal body			Cordovan body			Total
black	chartreuse	ivory	black	chartreuse	ivory	drones
184 (179)	187 (179)	352 (358)	159 (179)	191 (179)	359 (358)	1432

*Numbers in parentheses represent expectation on the basis of independence of the three loci. χ^2 test indicates no significant difference.

Summary

1. A genetic analysis of five independently occurring eye color mutants and one body color mutant in the honey bee, *Apis mellifera* is reported.
2. All mutants are recessive.
3. An allelic series of three eye color mutants, the chartreuse locus, was detected.
4. A modifier gene which appears to affect only the *ch*^{1} mutant is described.
5. No linkage between any of the non-allelic mutants was found.

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Cryptorchidism in Cocker Spaniels

Terry Pullig*

Many breeders believe that neither monorchidism nor cryptorchidism is inherited but is rather the result of a diet deficiency. On the other hand, the author has the female C which has produced a cryptorchid in each of her two litters; the first litter was sired by the male 30 which was closely related to her, while the second one was sired by the unrelated male 6. A letter from the manager of the kennels, in which male 6 and his sire are located, stated that male 5 has sired a monorchid since they have had him.

According to Koch,[1] cryptorchidism is inherited in many breeds. He further states that the descent of the testes is a simple dominant to cryptorchidism in breeds having pronounced head shortening and screw tail; apparently, cryptorchidism was caused by a defective functioning of the anterior lobe of the pituitary. McPhee, *et al.*[3] found several characteristics which appeared among the Poland China inbreds which suggested the segregation of recessive genes. These included color, cleft palate, scrotal hernia, and the ridgeling characteristics. Concerning the ridgeling characteristics, the authors had unpublished data which strongly indicated that it was inherited. However, the data were insufficient to show how many genes were involved.

Manning[2] used an experimental diet of spray-dried egg white at a 66 percent level as the sole source of protein for male albino rats. On this diet the testes failed to descend, or if already descended, they returned to the abdomen.

The first litter which was whelped on May 5, 1951, consisted of two males and five females. One male and two females died the

(*Continued on page* 264)

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ALLELIC AND LINKAGE RELATIONSHIPS OF FIVE MUTANT GENES IN HONEY BEES

(*Apis mellifera* L.)

WALTER C. ROTHENBUHLER, JOHN W. GOWEN AND O. W. PARK*

THE honey bee was the organism involved in what was probably the first hybrid segregation ratio ever published.[11] In August 1854 — 11 years before Mendel presented his paper to the Brun Society of Natural Science — Johann Dzierzon,[1] a Silesian beekeeper and theologian related that a hybrid queen bee derived from a cross of the German black race to the Italian yellow race produced drones in a ratio of one black to one yellow. Zirkle advances the idea that this ratio may have suggested to Mendel the desirability of progeny counts. Mendel himself bred bees, was interested in race crosses of them, and, it may be supposed, was interested in comparing his results from the pea experiments with data from bees. The results Mendel may have had from bees are not known.[2]

Even though bee genetics had an early start, almost no genetic work on bees followed the initial advance until about three-quarters of a century had elapsed. The primary reason for this unproductive period of time was lack of a means to control mating of bees. They mate only while in unrestricted flight. In 1927 Watson[9, 10] demonstrated a technique of artificial insemination of queen bees which provided definite control of mating and which soon was duplicated by others. Michailov[5] used this technique almost immediately to analyze the genetics of a mutation for white eyes in honey bees. Nevertheless a small number of successful inseminations and a large number of failures was the rule for several years. By 1945 modifications and additions to the Watson technique, principally by Nolan,[6] Laidlaw,[3] and Mackensen and Roberts,[4] made successful a great majority of attempted inseminations.

Considerable effort has been expended in attempts to gain understanding of honey-bee genetics as revealed by the more than 100 references listed by Ruttner and Mackensen[8] in their excellent review of honey-bee genetics. Much of this work has been of a biometrical nature as in studies of racial variation, or else has been information gained from breeding programs in which a degree of mating control was obtained by use of attempted isolated mating. Important work has been done in these ways, but as is also apparent in the aforementioned review, very few characteristics have been established as associated with a given gene.

The present report (data presented at the 1952 meeting of the Genetics Society of America[7]) is concerned with the genetics of five mutations in bees—three of them for white eyes, one for chartreuse eyes, and one for Droopy wings.† The three whites are desig-

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†The ivory stock was acquired from Dr. Lloyd R. Watson, Alfred, New York; cream from the Polhemus Apiaries, Ames, Iowa; snow from Mr. Herald Partello, Boone, Iowa; chartreuse from Mr. Harvey Walker, Urbana, Illinois, through Mr. Henry C. Dadant of Hamilton, Illinois; and Droopy from Mr. Homer Van Scoy, Candor, New York, through Dr. William Coggshall, Ithaca, New York. Sincere appreciation is expressed to everyone associated with our acquisition of these stocks.

NORMAL AND DROOPY-WINGED BEES

Figure 11

Comparison of wild-type (left) and droopy bees (right). A droopy bee using legs only, takes a few seconds to regain normal position after being placed on its back, whereas a wild-type bee regains normal position immediately with the aid of its wings.

nated ivory (*i*), cream (*cr*), and snow (*s*). These mutations for white eyes are phenotypically so similar that one cannot be certain of a visual separation of them. Eyes of newly emerged chartreuse bees are yellow-green, but become reddish-brown as the bee ages.

Table I shows the allelic relationships of these mutants. Every mutant was put into combination with every other mutant. In the first case two ivory queens were inseminated with semen from cream drones, and from these matings 1180 black-eyed (wild type) worker bees (diploid females) and no mutant worker bees were counted. Every other combination of mutations produced worker bees that were all wild type. In most cases additional matings were observed to produce only wild-type worker bees but actual counts were not made. From Table I it may be concluded that no mutant in this group is allelic to any other mutant of the group and secondly that each one is recessive to wild type.

The fifth mutation, called Droopy (*D*), affects wing position. Bees showing this mutation carry their wings spread from the longitudinal axis of the body and drooping from their point of attachment (Figure 11). Although the wing itself appears to vary but little from normal, Droopy bees cannot fly or use their wings to right themselves when placed on their backs on a flat surface. Their abdomens often are distended with feces accumulated because of inability to take cleansing flights.

Data revealing the genetics of Droopy are outlined in Table II. The original queen in the first generation was wild type in appearance, but produced Droopy and wild-type workers in a 1:1 ratio. The assumption most likely to be true is that this queen was heterozygous for Droopy in only her germinal line. In the second generation the original queen's Droopy daughters which were mated to unrelated wild-

TABLE I. Allelic relationships of four eye-color mutations

Number of queens	Genotype of queens	Genotype of drones	Wild type females	Mutant females
2	*i/i*	*cr/*	1180	0
1	*i/i*	*s/*	853	0
3	*i/i*	*ch/*	2282	0
1	*s/s*	*cr/*	228	0
1	*s/s*	*ch/*	190	0
1	*cr/cr*	*ch/*	109	0

TABLE II. Genetics of Droopy

Generation	Phenotypes of parents	Number of queens involved	Progeny D♀	Progeny +♀	Progeny D♂	Progeny +♂
1	+♀ × Unknown ♂ or ♂♂	1	138	132	none*	all*
2	D♀ × +♂♂ from non Droopy lines	1	243	242	---	---
		1	232	227	---	---
		1	some*	some*	0	449
		1	some*	some*	0	296
		9	some*	some*	---	---
2	+♂ × +♂♂ from generation 1	10	none*	all*	---	---
3	D♀ × +♂♂ from non-Droopy lines	7	535	487	---	---

* Based on observation, counts not made.

type drones produced Droopy and wild-type workers in a 1:1 ratio, whereas the wild-type daughters which were mated to drone progeny of the original queen produced only wild-type workers. A third generation of Droopy queens reared from second generation stock again produced worker progeny in a 1:1 ratio.

The situation among the drones is not so clear. No Droopy drones have ever been seen. It may be supposed that the Droopy mutation in the haploid drones is either lethal or unexpressed. Preliminary data from two Droopy queens on percentage of unfertilized eggs that develop into mature drones suggest that Droopy is lethal in drones.

From the data presented it is concluded that the Droopy phenotype is the expression of a single dominant mutation. It is probable that the Droopy mutation was present only in the germinal line of the original queen. Droopy seems to be lethal in drones.

Table III shows the linkage relationships of all five genes. Each one was tested with every other one. In the first case three queens heterozygous for chartreuse and ivory produced drones (actually gametes) in a 1:2:1 ratio. The ivory-chartreuse class is confounded with the ivory one since ivory is epistatic to chartreuse. The genes in question segregated independently. In matings from which worker bees were counted, the doubly heterozygous queens were mated to doubly recessive drones, which made it possible to count each of the queen's gametes in its own class. Every gene segregated independently from every other gene in the test.

Summary

Ivory (*i*), cream (*cr*), snow (*s*), chartreuse (*ch*), and Droopy (*D*) behave as monofactorial characters—the first four recessive to wild type, the last one dominant to wild type. Apparently five different non-linked loci are marked by these mutations.

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TABLE III. Linkage relationships of five mutations

Number and genetic constitution of queens tested	Gen. Const. of ♂♂ mated to ♀♀	Sex of Progeny Counted	Classification of progeny												χ^2	P	Conclusion
3 *ch;i*⁺/*ch*⁺;*i*	†	♂	107	*ch;*	198	*i* & *ch-i;*	96 +								0.7	0.7 -0.8	
2 *ch;cr*⁺/*ch*⁺;*cr*	†	♂	1019	*ch;*	2065	*cr* & *ch-cr;*	954 +								4.2	0.1 -0.2	
1 *ch;s*⁺/*ch*⁺;*s*	†	♂	198	*ch;*	392	*s* & *ch-s*	214 +								1.1	0.5 -0.7	
2 *ch;D*⁺/*ch*⁺;*D*	†	♂	376	*ch;*	0	*D;*	0 *ch-D;*	369 +							0.1	0.7 -0.8	
2 *i;cr*⁺/*i*⁺;*cr*	†	♂	903	*i,*	*cr,*	& *i-cr;*	300 +								0.003	0.95-0.98	No Linkage
1 *i;s*⁺/*i*⁺;*s*	†	♂	213	*i,*	*s,*	& *i-s;*	56 +								2.5	0.1 -0.2	
2 *i;D*⁺/*i*⁺;*D*	*i;D*⁺	♀	84	*i**;	87	*D;*	89 *i-D;*	84 +							0.2	0.95-0.98	
1 *cr;s*⁺/*cr*⁺;*s*	†	♂	124	*cr,*	*s,*	& *cr-s,*	48 +								0.8	0.3 -0.5	
2 *cr;D*⁺/*cr*⁺;*D*	*s;D*⁺	♀	58	*cr;*	52	*D;*	57 *cr-D;*	55 +							0.4	0.9 -0.95	
3 *s;D*⁺/*s*⁺;*D*	*cr;D*⁺	♀	98	*s;*	130	*D;*	120 *s-D;*	108 +							5.1	0.1 -0.2	

* Single gene symbols, in the cases of these worker bees, indicate the phenotype of the individuals.
† Not relevant.

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DOMINANT INHERITANCE OF RADIAL HEMIMELIA

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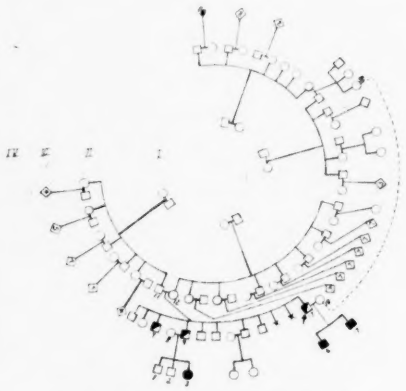
THE condition described in this paper is identical with that which was analyzed by O'Rahilly[6] and is called radial hemimelia. It was first described by Petit.[7] Radial hemimelia is a relatively frequent deformity with various degrees of expression. It has been reported in the literature by several authors. Kato[5] collected 253 cases up to 1923 and since that date many more instances have been described. Kato[5] makes reference to three authors who described this condition, and who reportedly stated that hereditary tendencies were found, but that this was not the usual but rather the exceptional. Kanavel[4] again states that hereditary tendencies have been found in some case histories but are apparently not common. He mentions Blencke[1] who reportedly found the condition in four children out of a family of nine. Kanavel[4] also cited Joachimsthal[3] who reportedly found a mother and five children with the same condition. It is worthy of note that these brief references to heredity do not indicate that a dominant type of inheritance is unlikely.

Evans[2] in describing a rare case of radial hemimelia in a Tarascan Indian girl again mentions heredity as being a possible explanation for the occurrence of radial hemimelia, but does not suggest a mode of inheritance.

It is the purpose of this paper to report a pedigree in which a dominant type of inheritance is strongly indicated for radial hemimelia. No attempt is made to explain the phenomena so frequently observed in skeletal defects which cause genes to be expressed either unilaterally or bilaterally, or to different degrees in different individuals. The grosser expression of the proposed gene involves the lack of the radius, some carpal bones, and the thumb, while its milder expression involves only a partial lack of development of the radius causing a compensatory displacement of the hand to the radial side and the lack of some carpal bones as well as the lack of the proper joint between the thumb and the carpus.



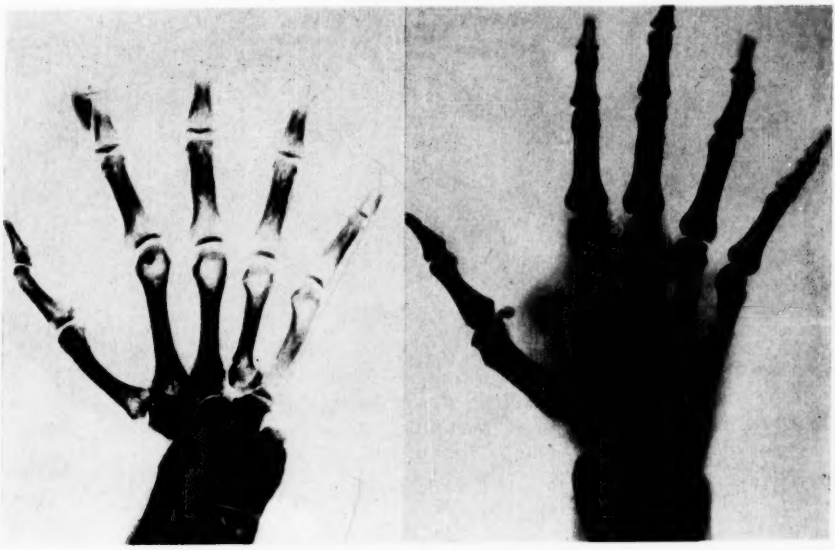
UNDER-DEVELOPED RADIUS

Figure 12

Pedigree of a family in which hemimelia is found. The milder expression of the gene is indicated by only half of the square being shaded while the grosser expression is indicated by the entire square or circle being shaded.

Materials and Methods

One pedigree, Figure 12, was collected in which the above mentioned anomalies occurred. These traits had not been evidenced in the ancestory of either the propositus III—17 or his wife III—18. Thirty blood relatives of III—18, going back two generations failed to show any signs of these traits. Ninety-eight blood relatives of III—17, likewise going back two generations, failed to show these traits. The only members of III—17's pedigree, besides his two affected children, IV—6, and IV—7 showing either of these traits, were his two brothers, III—7 and III—9, who showed the milder expression of the anomaly, and his brother's daughter, IV—3, who showed the grosser expression of the gene as thumblessness. III—17's two children IV—6 and IV—7 also showed the grosser expression of the gene.



AFFECTED AND NORMAL RIGHT HANDS

Figure 13

At the left is shown the skeletal elements of the right hand of affected individual, III-17, dorsal aspect. A normal right hand can be seen on the right.

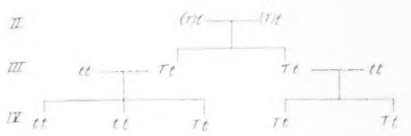
An analysis of the pedigree in an attempt to determine the fact and mode of inheritance was then undertaken by the pedigree elimination method.

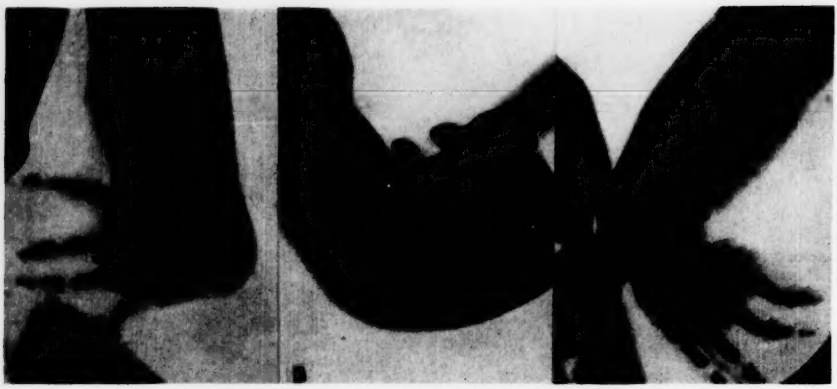
Results

The skeleton of the right hand of propositus III-17 is shown in Figure 13. The thumb of the left hand (not shown) is anomalous and fails to form a proper joint with the carpal bones. In appearance, the thumb is very slender and its function is greatly reduced. It is usually carried extended to the side and gives the appearance of being smaller in diameter compared to the other digits which are very well developed. There is also an absence of carpal bones evidenced, although the exact number is not clearly shown. There is also a marked displacement of the hand to the radial side to compensate for the hypoplasia of the radius as well as the lack of carpal elements and musculature of the thumb. There is an undifferentiated mass between the radius and ulna. The right hand of the propositus is affected but to a lesser degree. The right hand shows a functional thumb and all carpal bones present. This hand exhibits a lesser displacement to the radial side, but there is an undifferentiated mass between the slightly hypoplastic radius and the ulna. This condition is obviously similar to that of the other hand, but to a lesser degree. Similar conditions, in both cases showing a greater expression on one side than the other, exist in III—7 and III—9.

The skeletal elements in the hands of IV—7 are shown in Figure 14. Similar conditions exist in IV—3 and IV—6, in which total absence of both thumbs indicates a grosser expression of radial hemimelia than was found among the affected members of generation III.

A simple dominant gene with some lack of penetrance seems the simplest and most probable explanation. The chart below indicates the probable gene combinations. The dominant *T* gene is indicated here as having occurred in one of the parents of either II—11 or II—12 as a new mutation with a lack of penetrance in either II—11, or II—12. It was then passed on with varying degrees of phenotypic expression to their offspring. Modifying factors of one form





SKELETAL ELEMENTS OF RADIAL HEMIMELIA

Figure 14

The skeletal elements of the arms and hands of IV-7. *A* shows the left arm ventral aspect, *B* the left arm dorsal aspect and *C* the right arm ventral aspect.

or another must be present to explain these different phenotypic expressions. No other explanation is so readily applicable to the pedigree. Various other explanations and their applicability follow:

1. Multiple alleles are a second possibility here, but seem highly unlikely, as this explana- would have to assume that all three mothers, II—8, III—8, and III—18, of various ancestory brought in the proper allele.

2. All forms of recessive modes of inheritance seem unlikely also as they fail to fit the pedigree adequately.

3. Y-linkage or any form of sex-linkage also fails to fit the pedigree adequately, and are therefore likewise unlikely.

4. The possibility of a simple autosomal dominant causing thumblessness when homozygous and a milder expression when heterozygous is ruled out as all three mothers failed to give any expression of the gene.

Summary

1. Radial hemimelia is a fairly common deformity which has been frequently reported in the literature but with no satisfactory explanation of its hereditary aspects.

2. A pedigree was described in which radial hemimelia was found.

3. No attempt was made to explain either varying expression of the condition within an individual, that is, whether it was expressed unilaterally or bilaterally, or to different degrees of expression in different individuals.

4. A dominant type of inheritance was indicated, but the pedigree was explainable on this basis only if a reduced penetrance was assumed in one parent of the propositus.

5. Other forms of inheritance were discussed and shown to be unlikely.

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CYTOLOGICAL EVIDENCE OF HEXAPLOID CELLS

In Maize Endosperm

HOPE H. PUNNETT*

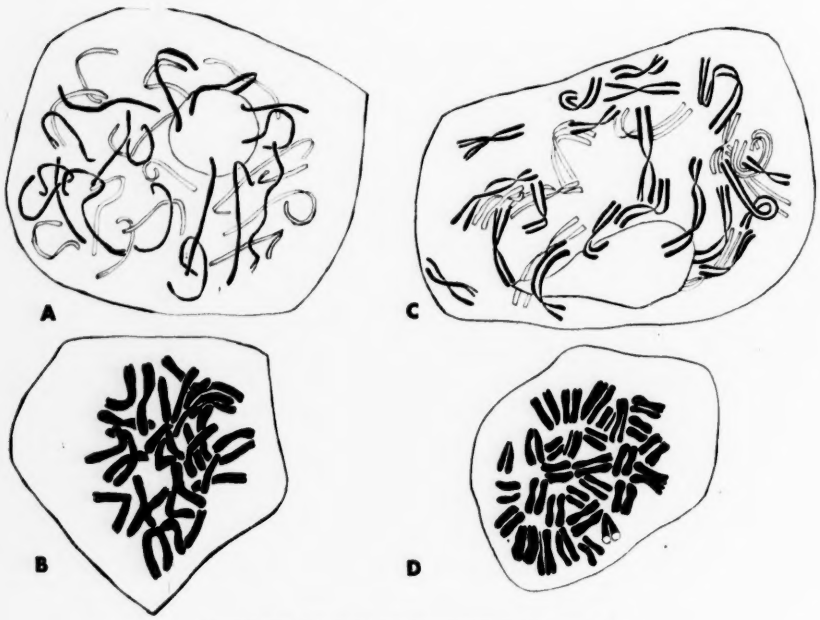
ENDOMITOSIS, the duplication of chromosomes without nuclear division, may be accomplished by three different methods: (1) repeated duplication of the chromonemata without subsequent mitoses, resulting in polytene chromosomes as found in specialized cells of *Culex* and *Drosophila;* (2) failure of spindle formation after a normal chromosomal duplication, resulting in cells with a doubled number of chromosomes such as the nurse cells of *Drosophila* ovaries; (3) occurrence of two duplications during the resting stage followed by a single normal mitosis as in *Spinacia* periblem.

In the third type of endomitosis the doubled chromosome number can be directly observed during the mitotic cycle. However, endomitosis of type one or two usually occurs in mature cells which have ceased dividing so that direct proof of polyploidy is difficult to obtain except in those insects where the chromosomes stain deeply enough to be counted in resting cells. In most plant and animal tissues, therefore, indirect indications of changes in chromosome complement must be sought. Duncan and Ross[1] in a recent paper on the differentiation of endosperm in *Zea mays* used nuclear volume and nucleolus and knob number to determine the degree of polyploidy in resting cells. Since the nucleolus in maize is associated with one chromosome in the haploid set of ten, triploid endosperm cells have three nucleoli. Cells with a multiple of three nucleoli may be assumed to have a corresponding increase in ploidy. (Nucleoli often fuse but the difference in size usually indicates the original number.) Duncan and Ross found no cells with more than three nucleoli. In the line of maize which they used, only two chromosomes of the haploid set possess knobs. A maximum number of six heteropycnotic bodies representing the knobs was observed in resting nuclei. Their evidence for endomitosis in corn endosperm is based on the increased nuclear volume of the large central cells and the number of strands attached to knobs in some energic nuclei. The authors stated that the latter resembled endoprophase in an endomitotic cycle but no other stages of such a cycle were ever seen.

Unlike Duncan and Ross's observation, 37 cells in all stages of a process resulting in hexaploidy were found by the writer while analyzing maize endosperm smears for evidence of a breakage-fusion-bridge cycle involving a fragment chromosome. The cells occurred singly or as sectors in kernels from three ears fixed in propionic alcohol eight days after pollination and stained as propionic-carmine or aceto-orcein smears. At this time little size difference exists among endosperm cells and, as also stated by Duncan and Ross, the nuclei are intermediate in size between the small peripheral nuclei and the large central ones which are differentiated within the next three or four days.

In the ten prophase cells observed, each of the 30 chromosomes appeared paired (Figure 15*C*). The members of each pair were double, as were normal prophase chromosomes (Figure 15*A*). The 21 cells found in metaphase were marked by persistence of the pairing and also by an apparently greater contraction than that found in nor-

*Work done while a National Science Foundation Predoctoral Fellow, Botany Department, University of Illinois. The author wishes to thank Dr. Marcus M. Rhoades for his advice and help.



MITOSIS IN MAIZE ENDOSPERM CELLS

Figure 15

Camera lucida drawings showing: *A*—Normal prophase; *B*—normal metaphase; *C*—prophase of endomitotic cycle with paired chromosomes; *D*—metaphase of same showing characteristic pairing.

mal metaphase plates (Figure 15*B* and *D*). In some late metaphase figures, the components of several pairs of chromosomes had become separated while those in other pairs remained closely associated. In three late anaphase cells 60 daughter chromosomes could be counted in each polar group. Two pairs of sister cells with five (two being fused) or six nucleoli per cell were found as well as a single cell with the doubled number of six. (Its sister cell had probably been lost during smearing.) No cells were observed in mitosis with 60 unpaired chromosomes and hexaploid cells were found in approximately half of the kernels examined in which division figures were present.

The conspicuously paired association of chromosomes described above is diagnostic for endoreduplication, a term introduced by Levan and Hauschka[2] to designate the third type of endomitosis. Since the events leading to the doubled chromosome number take place during resting stage when the chromosomes are despiralized, it is impossible to see any more within the nuclear membrane than during a comparable phase of normal mitosis.

De Litardière,[3] the first to recognize the significance of the paired somatic metaphase in the differentiation of *Spinacia* root, considered that two successive duplications had been interposed between the telophase of one mitotic cycle and the prophase of the next. Furthermore, the presence of cells with the 4*n* unpaired complement as well as with the 8*n* paired metaphases in spinach indicated that further divisions could occur normally after doubling and that the number of chromosomes could be redoubled in a further endoreduplicative cycle. In maize, the absence of metaphases with 60 unpaired chromosomes may indicate that hexaploidy is established in the last division before the cell becomes completely differentiated and meristematic activity ceases. No 6*n* figures were observed by the writer in kernels from ears fixed 10 to 14 days after pollination, nor were any observed by Duncan and Ross. If the endomitotic cycle is limited to the

division prior to maturation, both the low observed frequency of *6n* mitoses compared to *3n* and the absence of the former in many kernels may be explained.

Whether or not endoreduplication is the process regularly responsible for the large central nuclei is beyond the scope of this report. It is possible, as Duncan and Ross suggest, that multiplication of strands occurs after the cells have become differentiated. In this case, if endoreduplication is sporadic, only some cells would be *6n* by chromosome count but apparently all could have multi-stranded chromosomes. If this difference were reflected in nuclear volume it would be logical to expect a bimodal curve based on nuclear size. Unfortunately Duncan and Ross do not have sufficient numbers in their statistical plot of volume changes with age to confirm or deny this hypothesis. Only a quantitative cytological examination of early and late stages of development can resolve the question of whether or not endoreduplication is characteristic of the maturation of maize endosperm tissue. (It is not suggested that the time of sampling, eight days after pollination, is necessarily optimal for observing paired prophase and metaphase figures. The plants used were greenhouse grown and pollinated in mid-winter. Growth rates would undoubtedly differ for field-grown plants.)

Summary

All stages of an endomitotic cycle resulting in hexaploid cells were observed in eight day old maize endosperm tissue. That normal mitosis was following double duplication of the chromosomes during resting stage, was indicated by the presence of 30 somatic pairs of chromosomes in prophase and metaphase cells. Anaphase proceeded regularly. Sixty chromosomes could be counted at each pole and the daughter cells had the doubled number of nucleoli, 6. No further division of the *6n* cells was observed.

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WHAT IS MAN?

At the bar of justice, his life hanging on the answer, stands Douglas Templemore, journalist and thinker, lover of mankind, and father—for science's sake—of several offspring, possibly human. Derry, the strange creature who mothered the "son" that Douglas deliberately killed, is quartered happily in the London Zoo. Not science or Philosophy, not Parliament or clergy, could decide whether she is a manlike ape or apelike man. Yet the court must rule.

For if man can be defined, and if Derry is ruled to be human, Douglas has committed murder and must die. But if Derry is judged to be an ape, her whole species is condemned to legal slavery—precisely what Douglas is risking his life trying to prevent. Douglas thinks that he must lose, either way.

Such is the essence of the science fiction novel, "You Shall Know Them" by Vercors (Little,Brown). The book was written by Vercors, the chief literary spokesman of the French Resistance, and is an exciting tale, full of suspense and successive dilemmas.

The aim of this note is to consider the scientific possibility that a "missing link" like Derry might be found. Should a species of "tropis," as they are called in the book, be found, it would upset innumerable apple carts and give the delegates to the United Nations some first class headaches. The ability of the imaginary tropis to produce healthy offspring, when artificially, or naturally, inseminated with human sperm, would produce no end of problems in real life. According to genetic theory, such hybrids would be of rather inferior intelligence and only capable of performing the less intellectual tasks. Would they be admitted to labor unions or excluded with prejudice? It is unlikely that the hybrids would have sufficiently sophisticated attitudes to limit their reproduction voluntarily, so that it might be necessary to sterilize some of them in order to prevent overpopulation. Would there be religious objections to this?

The probability that such a titillating situation will arise is not worth mentioning. It is not likely that the tropis will ever be found, though probably similar organisms once did exist. A little speculation, such as that provided by Vercors, is worth the time it takes, because from it we learn to know ourselves

better. One might even speculate that by careful selection over some thousands of years for higher intelligence in chimpanzees we could produce a new race which would be able to adjust to human cultures quite successfully. At present the chimpanzee can be educated to do simple tasks and to enunciate a few words. They learn to love money and display all the emotions known to man.

Would this future super-chimpanzee be invited to compete on the gym team at the University of Minnesota? Just think how they could whiz through the air on the flying rings!

One serious point may be made. All known primates are much too distantly related to us to make the production of human-ape hybrids possible. Cross-breeding experiments with plants and animals have shown that, for the more specialized higher forms, only the extremely closely related species produce viable hybrids. The parents of the mule are different species, to be sure, but not fundamentally very different, and even with the mule there can be no pride of ancestry nor hope of posterity. With the passage of time, isolated species drift apart, reproductively speaking, and genetic barriers are set up which make future interbreeding more difficult and eventually impossible.

Let us return to the fantasy of the book. At the imaginary trial the jury could not reach agreement as to whether Derry, the tropi, is human or not, and discovered that what was needed was a definition of man—but no definition could be found. The jury was dismissed without reaching a verdict, and Parliament appointed a committee to phrase a definition of Man. The author decided (via the committee) that when an animal species evolves to the point where it has a spirit of religion, then it is human.

Vercors has a rather broad concept of what religion is, and consequently, of the nature of man. The tropis built fires and smoked their meat slightly, not because it tasted better but as an early manifestation of ritual cannibalism. The rites of cannibalism were considered an adequate sign of religion and therefore the tropis must be human and entitled to all the rights thereof.

The jury, at the new trial of Templemore, decided that, as the definition of man had not been available at the time he killed his "son" as a test case, he should go free. In the future the tropis were to be considered human.

This decision, that the tropis had souls, at least legally speaking, came too late to save the priest-anthropologist, who was on the expedition which discovered the village of tropis, from a great deal of soul searching of his own. He, too, could not decide whether they were men or beasts. If they were human, he must baptize them to save them from the eternal silence of limbo, if not the flames of hell. "On the other hand," he declared, "he didn't wish to find himself in the same boat with Saint Mael, whose eyesight was so poor that he mistook a tribe of penguins for a lot of gentle savages and baptized them forthwith. Now, how were the souls of penguins to be received at the right hand of God? A council of archangels decided that the only way out of the quandary was to change them into men. Which was done. Whereupon all those poor penguins ceased sinning in innocence, and were well and truly damned."

How can we determine when a soul has appeared in our super-chimpanzee of the future? History shows that religion has evolved slowly and painfully. In fact, so slowly, that only rarely could any change be perceived in one lifetime. Furthermore, not all questions of the relationship of the soul to the body in the present species of man have been resolved. Identical twins, for instance, start as one body and then divide into two. Does the soul divide into two at the same time? Apparently so, as both twins are christened. But every now and then the division may not be complete, with the result that there is one body and two heads. If the soul is located in the head, then there are two; if it is located in some other part of the anatomy, then there could be only one. Should this unfortunate monster be registered as one or two persons?

Scientists do not ordinarily spend much time cogitating about the soul. To the scientist, man is fundamentally a beast, as most women will agree; however, to the scientist, he is a special animal, in that he is considerably more intelligent than the others, and has a soul. Vercor's idea that his fictitious tropis are human, because they enjoy smoking their meat, seems to be somewhat of an over-simplified rudiment for the soul of man. The scientist prefers to leave the soul undefined and undefinable. However, the author, Vercors, is no scientist. He is just having some good clean fun by disturbing people.

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FRUITFULNESS, FERTILITY AND PARTHENOCARPY

In Tetraploid Species Hybrids of *Lycopersicon*

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SEEDLESS fruits which develop with or without pollination sometimes occur on the common tomato. These fruits usually grow very slowly and are smaller than seedy fruits. The loculi at maturity contain very little jellylike tissue. In a heterozygous deficient tomato[3] and in its tetrasomic progeny, the tendency to parthenocarpy of this type was much increased.

In a family consisting of four interspecific F_1 hybrids of *Lycopersicon esculentum* × *L. peruvianum* var. *dentatum*, three plants were diploid and one tetraploid.[4] In the field at Riverside, California, without controlled pollination, all produced a fair crop very late in the season. One season the tetraploid F_1 hybrid 42.90.1 fruited early and again very late. The tetraploid was more fertile than the diploids; its fruits were larger and usually contained six or seven seeds, only a few being seedless. The fruits of the diploids were usually seedless.

The F_1 tetraploid was backcrossed to an unusually fruitful, fertile tetraploid plant of *Lycopersicon esculentum*. In the first backcross generation 31 plants produced no fruit, two plants had only three or four fruits, and one plant was fairly fruitful. The most fruitful plant was selected, and after selfing for three generations, a second backcross was made to a small-fruited tetraploid *L. esculentum* derived from that used in the first backcross. Neither parent of this second backcross was parthenocarpic. In the two second backcross families 47.91 and 52.89, ten plants had fruits with many seeds, two had fruits with few seeds, and one had seedless fruits. Fruitfulness also varied widely and seemed to increase as the season advanced. In the shorter, warmer days of late July, about half of the plants had a good crop and the rest a light crop or none. On one of the more fruitful and seedy second backcross plants (52.89.2), 65 percent of the flowers set fruit, and on an unusually fruitful autotetraploid plant of *L. esculentum* var. Pearl Harbor, only 13 percent.

On the second backcross plant 47.91.7, practically every flower produced a fruit, and every fruit was seedless. The ovary was abnormally large prior to flower opening (Figure 16) and continued to grow to maturity as in a normal, fertilized flower. The fruits were solid, averaged 24 grams in weight, and had two or three loculi containing green pulp when immature (Figure 17). The skin, rind, and outer fleshy pericarp of the mature fruit were yellow, and the placenta and jellylike tissue surrounding the abortive seeds were orange tinged with red. The colors clearly indicated the presence of genes modifying flesh color from *L. peruvianum*. The foliage was more like that of *L. esculentum*, and the plant was distinctly lacking in vegetative vigor, perhaps because of its extreme fruitfulness and the presence of tobacco mosaic disease. Pollen from two *L. esculentum* tetraploids and a diploid was applied to buds and flowers of 47.91.7, but no seeds developed.

Open pollination of 47.91.5, a much less fruitful sib of 47.91.7 having red fruits, gave 11 seedlings, none of which was very fruitful. Eight plants had only seedy fruits or a mixture of seedy and seedless, one had seedless fruits only, and two plants had no fruit.

Pollen mother cells of 47.91.7 had 48

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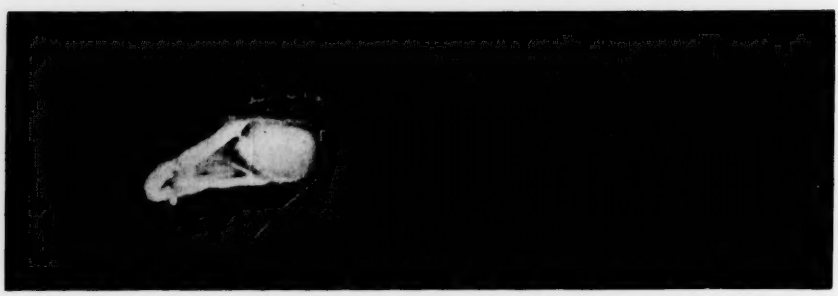


PRECOCIOUS GROWTH OF OVARY

Figure 16

Bud of parthenocarpic tetraploid *Lycopersicon* species hybrid 47.91.7, showing abnormal growth of the ovary before flowering. (Approximately 2½ ×.)

chromosomes, and the number of bivalents and tetravalents was similar to that in autotetraploids and in the original F_1. Meiosis was of the type expected of a normal tetraploid, but the young pollen stopped growing when it was about half the mature size, and then aborted, so that at maturity the plant was totally pollen sterile.

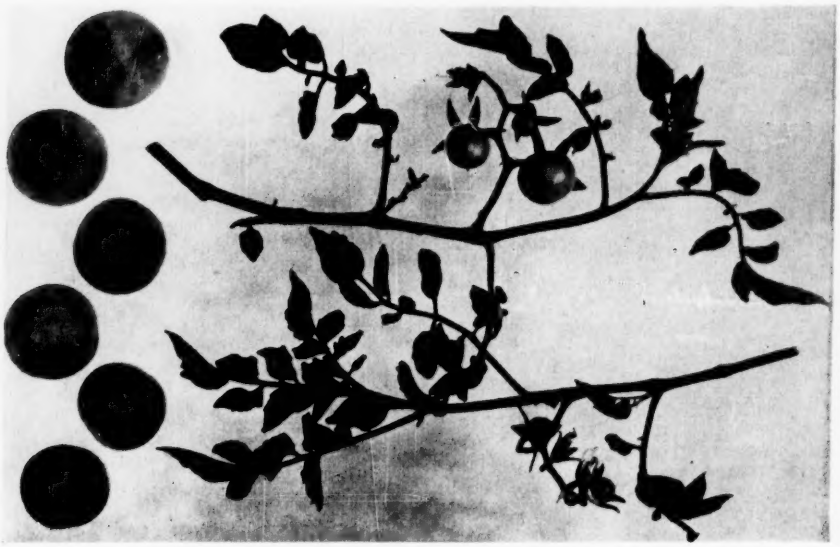
Cross sections were made through whole flowers of 47.91.7. A few megasporocytes had undergone cytokinesis, and several were in diakinesis, metaphase I or telophase I, while cytokinesis was beginning in the pollen mother cells. Cross sections through whole flowers of two other tetraploids and a diploid showed reduction occuring in embryo-sac mother cells when young pollen was present in the anthers. This may indicate that the megasporocytes of 47.91.7 develop somewhat prematurely. The embryo sac forms normally, but the cells of the endothelium surrounding it soon grow very large and tend to grow into the embryo-sac cavity, which is therefore much more variable in size and usually much smaller than in normal tetraploids. Rick[10] describes similar hypertrophy of integumentary cells in some embryo sacs of triploid tomatoes. The ingrowth of the endothelium also resembles the "ovular tumors" described by Rappaport, Satina, and Blakeslee[9] as occurring in incompatible crosses of *Datura*, and the intrusion of cells into the embryo sac that gives rise to nucellar embryos in citrus, described by Strasburger.[11]

Discussion

Autotetraploids of *L. esculentum* are less fruitful and less fertile than their diploid sibs, and do not tend to be parthenocarpic. The difference in fertility has been attributed to meiotic irregularities. Evidently, the tetraploid species hybrids of *Lycopersicon* described above are more fruitful and more fertile than these autotetraploids. This increase in fruitfulness and fertility was not associated with more frequent bivalent formation and reduced multivalent formation in meiosis. Meiosis in the pollen mother cells of the highly fruitful second backcross plant 52.89.2 was indistinguishable from that in autotetraploids of *L. esculentum*. The complete sterility of the extremely fruitful hybrid 47.91.7 indicates that fertility and fruitfulness may be dissociated.

Müntzing[5] came to the conclusion that the inferior fertility of experimental tetraploids, when compared with natural tetraploids, was due not so much to meiotic irregularities as to genotypic differences. Randolph,[8] working with *Zea mays*, and Müntzing,[6] with *Galeopsis*, found that hybridization of strains of the same species may give increased fertility. Westergaard[12] reported that in *Solanum* the fertility of amphidiploids from crosses between diploid parents depended on how far the genomes of the diploid parents can harmonize, rather than on the meiotic behavior of the amphidiploids. Some tetraploid intersubspecific hybrids of *Oryza* studied by Cua[1] were more fertile than autotetraploids of the parent subspecies.

The fact that seedless tomatoes similar to those of 47.91.7 can be made to develop without pollination[2] by applying auxins, suggests that an excess of some similar substance in the cytoplasm caused 47.91.7 to be parthenocarpic. Since both pollen and ovules fail to develop normally, and since the ovary grows unusually large before the flower opens, it



PARTHENOCARPIC TETRAPLOID

Figure 17

Every flower on the *Lycopersicon* hybrid 47.91.7 developed into a seedless fruit, but vegetative growth of the shoot was slow.

seems improbable that this substance is derived from either pollen or ovules, which, as Nitsch[7] points out, normally function in auxin formation and fruit production. Certainly, vegetative growth of the integument is at the expense of the developing megasporocyte.

The presence of a single partially parthenocarpic plant in family 47.91, and the predominance of semiparthenocarpic and non parthenocarpic plants in the progeny of 47.91.5, suggests that parthenocarpy may be due to an exceptional combination of genes from the two species, and that a tendency to parthenocarpy of the vegetative sort may be a large factor in yield of fruit and vegetables. The increased fruitfulness which frequently accompanies heterosis may in some cases be due to a factor similar to the one which caused that condition in the tetraploid. Species hybridization seems effective in increasing parthenocarpy in tomatoes.

The heterozygous deficient plant mentioned above suggests that the tetraploid parthenocarpic plant might have a similar deficiency in two chromosomes. In a diploid, this would be easy to determine, because the unequal chromosomes often fail to pair. The irregularities normally present in all tetraploids made this impossible to determine for 47.91.7.

Parthenocarpy of a less extreme sort than that of 47.91.7 occurred in diploids originating from another species cross, *Lycopersicon esculentum* × *L. hirsutum*, and is therefore not necessarily associated with tetraploidy.

Summary

A tetraploid plant that originated from the cross *Lycopersicon esculentum* × *L. peruvianum* var. *dentatum* was female and male sterile, nearly 100 percent fruitful, and wholly parthenocarpic. Growth of the ovary was not interrupted at anthesis. The same degree of fruitfulness did not occur in either parent species, in F_1, in the first backcross generation to *L. esculentum*, or in the three generations of inbreeding that followed. Another tetraploid hybrid of the second backcross was more fruitful and more fertile than autotetraploids of two very fruitful varieties of *L. esculentum*. Increased parthenocarpy also occurred in diploid plants of another species cross; hence it is probably not due to tetraploidy. The increased fruitfulness was not associated with increased chromosome pairing but probably with an exceptional combination of genes of both species, which cause excessive auxin production. The increased fruitfulness in heterosis may be due to a combination of genes which causes a physiological change similar to that which induced parthenocarpy in the parthenocarpic tetraploid hybrid.

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Cryptorchidism in Cocker Spaniels

(*Continued from page* 250)

first week. The remainder of this litter consisting of one male and three females lived to maturity. The second litter also consisting of two males and five females was whelped on September 26, 1951. Because of the fact that they were accidentally chilled during the first 24 hours of life, all died by the time they were ten days of age except one male and one female, both of which lived to maturity.

The diet of these two litters consisted of ground lean beef, evaporated milk, pablum, vitamin drops containing A, B, C, D and E, and a mineral mixture. At about three or four weeks of age a good dog meal was substituted for the pablum.

The male in the first litter was started on injections of testosterone at six months of age and continued until he was about eight months of age, but the male in the second litter was not given testoterone. Both males remained cryptorchids.

The diet which these dogs received was not deficient as the two dog meals fed had the minimum requirements for normal health and reproduction in dogs. The additions made were more than sufficient to prevent any diet deficiency.

These data indicate that cryptorchidism is inherited as a recessive sex-limited character to the normal descent of the testes in cocker spaniels. There are several questions which require more data in order to arrive at anything resembling a satisfactory explanation of cryptorchidism, such as: (1) Are monorchidism and cryptorchidism produced by the same gene, but with varying expressivity? (2) Are they produced by the same gene, but with modifying factors producing the variation? Or

RELATED FAMILIES WITH CRYPTORCHIDISM AND MONORCHIDISM

Figure 18

Two pedigrees of cocker spaniels showing inheritance of cryptorchidism and monorchidism as a sex limited character.

(3) are there two different alleles for the two conditions with cryptorchidism dominant to monorchidism?

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STREAKED HAIRLESSNESS IN HOLSTEIN-FRIESIAN CATTLE

A Sex-Linked, Lethal Character

FRANKLIN E. ELDRIDGE AND F. W. ATKESON

A HERD of registered Holstein-Friesian cattle in which several animals had abnormal hair-coats was called to the attention of the authors. The farm was visited twice, with about nine months elapsing between trips, and all of the animals, both normal and abnormal, were inspected. Photographs were taken of all affected animals, and pedigrees were obtained for all animals related to those affected. Feeding and management conditions were checked carefully as a possible cause of the anomaly.

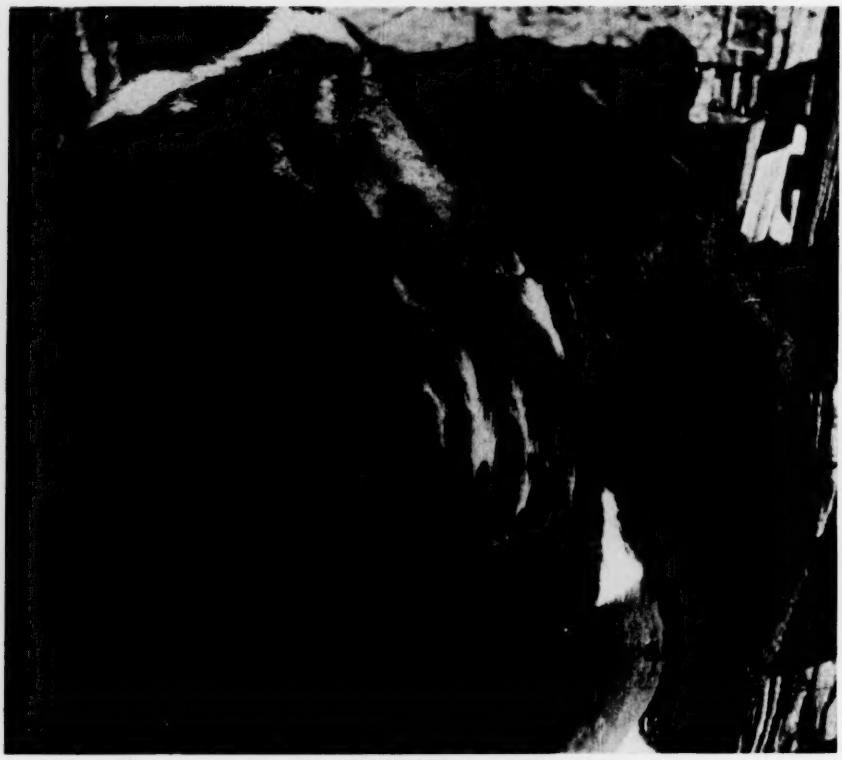
Description

The affected cattle were found to have areas devoid of hair on various parts of the body, the hairless areas occurring in more or less consistent patterns, (see Figures 19 and 21). On all affected animals there were approximately perpendicular hairless streaks over the thurls, some more extensive than others, with considerable variation between the two sides of the same animal. Hairless streaks also were found along the sides of some animals, being more extensive over the dorsal portion of the ribs than in the ventral areas. Also some hairless streaks were found on the legs, both above and below the hocks and knees. Each hairless streak was bounded by areas in which the hair-coat appeared to be rather sparse, although nearly normal in length. Hairless streaks occurred without regard to color pattern, both white and black areas being affected. The affected animals included some which were mostly white and some which were mostly black.

A large amount of variation occurred in the extent to which the animals were affected. Some were discovered easily since large areas of hairless skin were exposed to view, while other affected animals had such small areas that they had escaped detection by the owner. One of the mature animals, (II-1 in Figure 20) which was a daughter of an affected cow, and which also had given birth to an affected calf, previously had been considered normal by the owner. Close investigation of this animal at the time of the first visit to the farm revealed a small hairless spot on the right hip and some hairless skin near the ears. It was not feasible to catch the cow and inspect her minutely, but the appearance of the skin in the hairless patches and the fact that she was the only individual that did not fit into the pattern of heredity established by the remainder of the herd, left little doubt at that time that she was affected. Following the second visit to the farm, this cow, (II-1, Figure 20), was purchased and brought to Kansas State College for histological studies and at that time several other typical hairless areas were found and the generally sparse hair-coat noted. Clipping the hair-coat closely revealed several hairless streaks that could not be detected before clipping.

One six-weeks-old calf which looked exceptionally rough (IV-19, Figure 20), was found on close examination to have a number of streaks without hair, each streak overlaid by hair from the adjoining area in such a way as to leave no hairless areas readily visible. Following the discovery of this affected young calf,

*Contribution No. 221, Department of Dairy Husbandry, Kansas Agricultural Experiment Station, Manhattan, Kansas, as a part of the North Central Regional Cooperative Dairy Cattle Breeding Research Project.



DETAILS OF HAIRLESS STREAKS
Figure 19
Most of the skin is very smooth, but dorsally some scabbiness develops which is probably due to more direct exposure to the sun. Another affected cow can be seen in the background, upper left.

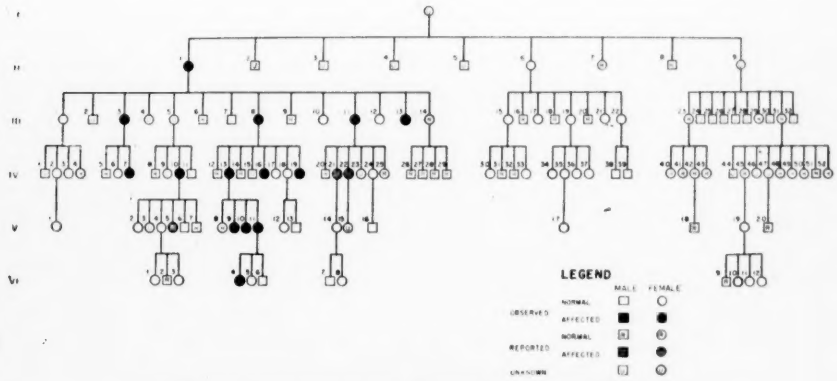
all of the other calves were scrutinized closely and were found to be normal. Thus the characteristic was found to be one that can be identified at an early age upon close examination, and suspicious animals can be separated from normals by the typical pattern of roughness of the pelage.

The hairless skin was smooth, wrinkled easily, and upon superficial examination, appeared to be thin. On one of the predominantly white animals and on another which was mostly black, there was considerable *scabbiness* on the hairless areas of the back. However, the hairless areas on the sides of these animals were smooth and free from scabbiness, indicating that the scabbiness was probably the effect of exposure of the skin to the sun and not a primary response of the trait itself.

Although large numbers of cattle have been observed by the authors, no other cases precisely like these could be recalled. It was observed, however, that the appearance of the hairless streaks could be mistaken easily for areas in which a cow had recently rubbed off the hair. Under circumstances where only one such animal was found, or where relationship was not established between animals, the ocurrence of these streaks would cause no one to suspect it might be a heritable character.

Effect on Cattle

The cattle were examined first on a relatively mild winter day and the authors were not able to observe personally the effect of



FIVE GENERATIONS OF STREAKED HAIRLESSNESS
Figure 20

In this pedigree chart only direct descent through females is shown. Cow II-1 was the oldest observed animal with streaked hairlessness.

cold temperatures on the affected animals. The owner stated, however, that the anomalous cattle were much more sensitive to extreme cold than normal cattle, that they would hump up and start shivering much sooner than the cattle with complete hair-coats. He also stated that when washing one of the affected heifers in preparation for showing as a 4-H project they found the skin to be more sensitive to the scrubbing brush than seems to be true of normal cattle.

Inheritance

At the time of the first inspection of the herd by the authors, the owner already had identified the fact that this anomaly was occurring in only one line of breeding in his herd, and that the affected animals were "usually" from affected dams. As the pedigrees of the affected animals were assembled these observations were verified. All affected animals were direct descendants of one prolific foundation cow born in 1926. In order to determine ratios of normal and abnormal animals among the progeny of certain types of matings, and to find an explanation for the apparently hereditary transmission of the characteristic, observations by the authors or reports from the owner were recorded for all of the offspring descending directly from this old cow for all generations up to the time of the inspections. This information was arranged on a pedigree chart, Figure 20.

Although the preliminary analysis of the data indicated that the characteristic might be inherited as a single dominant, perhaps sex-modified, the possible contribution of the males in the herd needed to be examined. During the years in which the streaked hairlessness had been occurring, five different bulls had been used, each of which had sired at least one anomalous heifer calf. The highest coefficient of relationship between any two bulls, as calculated from five generation pedigrees, was found to be 8 percent, and the average of all combinations of pairs was only 2.33 percent. For the characteristic to have been inherited as a recessive would have required the assumption that each of the five bulls was a carrier, or heterozygous. For each of the bulls to have been heterozygous would have required a high gene frequency in the breed and in this herd, since the five bulls were no more closely related than would be expected among a sample of five bulls picked at random from the breed. Only one bull was kept at a time for breeding purposes and each bull was kept for about three years, so there was little chance for selective matings to have disturbed the ratios. Also the characteristic has not been distributed widely throughout the breed, as stated previously. These combined facts practically elim-

TABLE I. Sex distribution of calves in pedigree shown in Figure 2

	Males	Females	Chi-square based on 1:1 hypothesis (Yate's correction used)
All Calves	47	78	7.20†
Calves which were direct descendants of II-1.	23	44	5.97*
Calves from remaining females.	24	34	1.40
Calves from affected descendants II-1.	12	34	9.59†
Calves from normal descendants of II-1 (excluding unknown III-5).	9	8	

* Significant (Probability $< 0.05 > 0.01$)
† Highly significant (Probability < 0.01)

VARIATION IN STREAKS
Figure 21

A—The approximately vertical streaks are found along the entire length of the body, especially noticeable in the dorsal region. B—Shows hairless streak on the foreleg, which extends from near the elbow point to below the knee. *C*—Shows a severe case of streaked hairlessness. This cow was the most severely affected of any observed, being nearly devoid of hair over the thurls. *D*—Shows the streaked hairless condition in a calf. The typical roughness of the coat which distinguishes the affected animals from the normal calves can be seen in this calf.

inated the possibility of the trait being inherited as a recessive, and at the same time absolved the five bulls from responsibility in the transmission of the characteristic. Therefore, data on these bulls were not included in the pedigree chart, Figure 20, since such data would have complicated the chart considerably without adding any useful information.

Since it was not possible for the authors to observe all of the animals which had been born in the herd since the characteristic was first discovered by the owner, the use of some of his observations increased the data available for study. Owner identification of the abnormal animals was considered to be quite reliable since he was familiar with the characteristic and would be inclined to positively identify as abnormal only those animals which were unmistakably affected. On the other hand, the accuracy of the observations of the owner concerning the normal animals was more questionable since he was not making a critical examination of each animal with regard to the presence or absence of the hairless streaks. Some animals only slightly affected or animals disposed of at early ages could have been reported erroneously as normal. Every effort was made by the owner to be as unbiased as possible and to report only those facts of which he was certain. Other members of the owner's family entered into the discussions and when they could not agree the animal was recorded as unknown.

Up to the time of the visits, there had been no attempt on the part of the owner to select against the defect, and apparently there was no natural selection against it since the oldest animal known to be affected was 15 years old and had 13 calves while the next oldest animal was 11 years old and had eight calves. Consequently there was probably no disturbance of the ratios of normal to abnormal progeny. Including only the animals observed by the authors, there were 10 normal and 11 affected females born to affected dams, a very close fit to the expected ratio of 1:1 from heterozygous females mated to homozygous recessive males. When all animals reported by the owner were included, in addition to those observed by the authors, there were 13 normal and 15 affected females born to affected dams.

The three normal females, III-1, IV-18, and

V-4, even though from affected dams, have founded small sub-lines in which all animals have been normal.

Two sons of affected dams, III-9 and IV-15, were sold into neighboring grade herds of cattle. Although accurate records of breeding were not kept, neither of these herds has any animals which were even considered suspicious of having streaked hairlessness. One of these herds was visited and the daughters were inspected. No affected cattle were found. No abnormal sex ratio was reported in either case.

Early in the investigation it was noted that the affected animals were limited to females. Since the numbers were rather small, the male calves usually were sold for veal at an early age, and the characteristic was found to be more easily identified in mature animals than in calves, the simplest and most obvious explanation of the absence of affected males at first appeared to be a lack of critical observation. However, it was also noticed that the sex ratio of these cattle through the female line of ancestry, as shown in Figure 20, deviated from the normal sex ratio. In the entire family descending from I-1, Figure 20, there were 78 females to 47 males, the difference from 1:1 being highly significant when tested by the chi-square method. This deviation was caused by the offspring from II-1, Figure 20, whose descendants had a sex ratio of 44 females to 23 males, also highly significant as tested by the chi-square method. The remaining animals descending from I-1, Figure 20, had a sex ratio of 34 females to 24 males, statistically a non-significant deviation from 1:1 although in the same direction. The descendants from II-1 then were divided into two groups, those whose dams were reported or observed to be affected and those whose dams were reported or observed to be normal. (The offspring of III-5 were not included since her condition was unknown, even though the occurrence of affected offspring pointed toward her being a carrier and therefore probably affected.) The offspring from normal dams had a sex ratio of eight females to nine males. The offspring from affected dams had a sex ratio of 34 females to 12 males which is not significantly different from the expected ratio of 2:1 for a sex-linked lethal gene.

If streaked hairlessness were caused by a sex-linked, lethal gene, the calving interval for affected cows should be longer than for their normal herdmates, since one-fourth of the pregnancies initiated would terminate in the death of the zygote. The difference in length of calving interval would depend upon the stage at which the zygote died. Since no aborted fetuses or unusual breeding behavior had been noted by the owner from the affected cows in comparison with the normal cows, it was assumed that the death of the developing zygote must have occurred in the early stages of pregnancy. Fortunately this was a herd of registered cattle in which accurate breeding records were kept. The average of the 46 calving intervals for the normal half sibs was 377 ± 6.5 days, and the average of the 39 calving intervals for the affected cows was 392 ± 6.9 days, 15 days longer. The variation in length of calving interval in this herd, however, was great enough that the difference of 15 days was not significant statistically on this small sample. Since no record was kept of the dates on which the cows were bred but did not conceive it was not possible to determine whether there was any greater tendency for the affected cows to skip a heat period than for the normal cows. Furthermore, with the variation which was present in the calving intervals it was not considered justifiable to attempt to establish the time of death of the embryo any more accurately than to state that it apparently occurs very early in its development. There was no significant difference between the lengths of gestation periods of the two groups of cows.

The above-mentioned facts may be summarized as follows: (1)—all affected animals were females and were descended directly from one female, (2)—each of five unrelated bulls sired one or more anomalous calves, (3)—the ratio of normal to abnormal females from affected dams bred to normal sires approximated closely the 1:1 ratio, (4)—only normal calves were born from normal dams, even though the dams were from affected families, and (5)—the sex ratio of calves from affected dams was close to two females to one male. These facts seem to justify the conclusion that the characteristic of streaked hairlessness is the result of a single, sex-linked, partially dominant or semidominant, lethal gene.

Discussion

Many variations occur in the completeness of the hair-coat of individuals from many different species. The most striking of these variations is probably the complete hairlessness, or nearly complete hairlessness that has been reported as occurring in mice, horses and dogs as early as 1894 by Bateson.[1] Other cases of nearly total hairlessness in several species were reviewed by Mohr and Wriedt[7] with special attention given to the hereditary aspects. In cattle, a number of investigators[2, 4, 8, 9, 10] have reported hereditary cases of hairlessness, of which some types are lethal. It may be that several of these reported cases of hairlessness in cattle are due to the same gene but the lack of cross-matings makes such an assumption inconclusive.

Two other types of deficient hair-coat in cattle have been reported. Cole[2] reported a case of an affected sire transmitting to some of his calves a short hair coat accompanied by defective teeth. He did not specify the mode of inheritance. Craft and Blizzard[3] reviewed the reported cases of hairlessness in several species up to 1934, and reported a new characteristic which was inherited as a simple reces-

sive, and which they called "semi-hairlessness." The "semi-hairless" characteristic was manifested by a thin coat of short, fine, curly hair at birth which started to change within two or three weeks to hair of a coarser type. The hair-coat of these animals remained coarse, wiry, and thin as the calves matured and they also appeared to be wilder than normal calves from the same breeding.

The cases of streaked hairlessness described in this report are not only different from any other cases known by the authors to be reported in cattle, but are different from any reports discovered by the authors about other species.

Although the animals with the hairless streaks were reported to be more susceptible to the cold than normal animals, this susceptibility apparently had not resulted in a decreased viability under the prevailing conditions. It is conceivable that such a characteristic might be an economic hazard for cattle kept under more rigorous conditions. It was roughly estimated that not more than 5 percent of the hide was lacking in hair in the most severely affected animal, and no single, hairless streak had a total area of more than about eight square inches. The combination of this relatively small hairless area, and the partial covering resulting from the alternation of hairless and approximately normal streaks might have alleviated to some extent the apparent susceptibility of these animals to cold.

Within the scope of the authors' knowledge streaked hairlessness, as reported herein, is the first character to be reported in dairy cattle for which the causative gene can be quite definitely located on a specific chromosome. Furthermore, it is the first characteristic which has been reported in dairy cattle to be associated so positively with a modified sex ratio.[5] It is still possible that further investigation will lead to different conclusions but this is not highly probable in light of the numbers already observed.

Genetically the occurrence of the abnormal sex ratio and the lack of affected males could be either a pleiotropic effect of the gene causing streaked hairlessness, or result of a gene closely linked to it. Without the phenotypic character to separate the carriers from the non-carriers, a sex-linked lethal gene easily could go undiscovered in cattle due to the relatively small number of offspring from one female. Only by combining the data on sex of offspring from several affected cows has it been possible in the case of streaked hairlessness to demonstrate a statistically significant deviation from normal.

So far as has been observed to date, the characteristic does not seem to be especially detrimental, although the lack of a complete hair-coat certainly is not a desirable trait. However, it might be that the increase in heifer calves in proportion to bulls, with apparently little loss in breeding efficiency, might be of sufficient importance or value to justify maintaining the characteristic. Hauschka, *et al.*[6] found a sex-linked lethal in mice that in the heterozygous condition was actually beneficial, the size of litters from such females equalling or exceeding the litters from normal females when a 25 percent smaller litter size would be expected theoretically. Such an explanation could possibly account for the apparent absence of any detrimental effect of the streaked hairless gene on the fecundity of the carrier cows.

A breeding program designed to take advantage of this sex ratio would be rather slow since transmission is only from female to female. The 2:1 sex ratio, (a 17 percent increase in heifers) would allow for more rapid building up of herds, or for a greater culling rate of females after the herd was established. Certainly one bull out of three calves is sufficient to keep reproduction going. New problems most certainly would present themselves in ways not now predictable if such a program were attempted. Also, at a time when a great cow had been found and a bull calf was desired there might be a great deal of objection on the part of the individual breeder since the ratio would still be effective.

From the standpoint of fundamental genetics, the gene for streaked hairlessness becomes a marker for one linkage group, and thus is the first step toward building a chromosome map for cattle.

Summary

1. An abnormality of the hair-coat in which approximately perpendicular, irregularly narrow streaks of hide on various parts of the body are devoid of hair has been observed in 13 females and reported on four additional females, all 17 affected animals descending directly from one cow in a herd of registered Holstein-Friesian cattle. Insofar as can be discovered by the authors this condition has not been reported previously in cattle nor has a similar condition been reported in any other species of animal.

2. No males have been found with streaked hairlessness.

3. An abnormal sex ratio in the family of cattle was the direct result of the ratio of 34 females to 12 males born to affected dams, a ratio not significantly different from the 2:1 ratio found with sex-linked lethals.

4. It was concluded that streaked hairlessness was inherited as a semidominant, sex-linked lethal.

5. The condition of streaked hairlessness reportedly causes the affected animals to be somewhat more susceptible to extreme cold than normal animals.

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"The O-Bomb"

MOZART, e.g., when asked by an admirer to tell the secret of his genius, shrugged his shoulders, and all that he could offer in explanation was that symphonies emerged when he seated himself before a keyboard; a rather complex response to a simple situation!

Some years ago I had the pleasure of admiring a musical genius of St. Louis. He carried our upright piano, mounted on his back, down three flights of winding stairs in the rear of our apartment. When he came back for more, I said, "George, how in the world can you do it?" And he answered, "Oh that's nothing, General, all you have to do is eat a-plenty." Nevertheless, those who witnessed this performance saw more than brute force. It was genius.

So here we have the wisdom of the body in the head of the femur, and the wisdom of the mind in the genius of Mozart and George.

The duplication of the miscellaneous wisdoms of the body by the brain of man is, to me, one of the most interesting phenomena of nature. It would seem very near the truth that an intellect has arrived virtually capable of comprehending nature if spiritual values are excluded.

The impact of the power of the intellect, and the relative roles of the wisdoms of the body and of the mind have, by strange circumstance, posed the great problems of the day.

Puny in body, deficient in tooth and claw, inferior in strength and speed, lacking in fur and armor and venom, man has duplicated all of these gadgets of living *for himself by force of intellect.* The speed and streamlining of the mackerel; the jet propulsion and smoke screen of the squid; the echo location of the bat; the physical power of the rhinoceros; all of which were built into the *substance* of these creatures by hundreds of millions of years of evolution, have within recent years become available to man as artificially acquired accoutrements of living and of fighting. These marvelous and wicked gadgets, crowned by atomic and hydrogen bombs and ingenious biological agents of death, have been made available to the unprepared emotions with unexpected suddenness.

The dangers of these instruments of destruction are presently uppermost in the minds of the world, but a vastly greater, a far more insidious danger threatens. An overpopulated planet, unable to offer respectable subsistence to its present inhabitants, is forced to provide for an ever-increasing number of guests. Man is increasing at the astounding rate of 68 thousand per day, or 25 million a year, literally a biological explosion. Overpopulation is sometimes referred to as the "O" bomb. In the effects which it produces on the human race, it is indeed the most explosive of the man-made bombs. Each year, 25 million newcomers, requiring at least 25 million acres of arable land for their subsistence, are forced through no fault of their own, to compete for what rightfully belongs to the millions already on the scene. *This is population pressure in its simplest terms.* Yet you and I, still enjoying the comforts of a rich land, scarcely appreciate the stupendous drama that is unfolding.

Were Thomas Austin here he could relate how easily ecological balances are upset, for it was he who imported 24 wild rabbits into Australia in 1859. The rabbits had the better of it for a time, for they had arrived in the island of Utopia of no predators. They were truly on the dole, and as they gave vent to their powers of reproduction they overran Australia, and millions and millions were to perish at the hand of man. It is obvious that neither Austin nor the rabbits knew what they were about.

When medicine, on the other hand, vanquishes one predator of man after another,

which is nothing more than Rabbit Utopia by scientific approach, it is presumed that science knows what it is about. When medical research reaches a proficiency which it cannot use wisely and humanely, it would seem time to call a halt to randomness of medical research until moral obligations have caught up with technology. It does indeed seem likely that a transfer of present federal support of medical research to medical care, during present critical adjustment of emotions to medical technology, would prove to be an act of mercy.

India, like all other countries, is increasing in population; about 6 million per year. A Dean of a medical school, who visited me recently, volunteered the information that this increase was due to an "improvement of medicine." A Professor of Physiology, also from India, expressed to me the fear that the United States was about to repeat the mistakes of India. He felt very strongly that a reduction of the population of India to one-half the present numbers would be the greatest blessing that could be bestowed on his country.

Vogt has called attention to the population problem in Puerto Rico as follows: "In many parts of the world, doctors apply their intelligence to one aspect of man's welfare and deny their moral right to apply it to the problem as a whole. Through medical care and improved sanitation they are responsible for more millions living more years in increasing misery. Their refusal to consider their responsibility in these matters does not seem to them to comprise their intellectual integrity. They have been primarily responsible for making Puerto Rico, for example, one of the most miserable areas on the face of the earth by expanding population beyond all possible bounds of decent subsistence, and their present efforts to correct the situation are not much more than tokens. They set the stage for disaster; then, like Pilate, they wash their hands of the consequences."

Overpopulation is recognized to be the most urgent problem of man by authorities of the highest standing in biology. Osborn, in "Our Plundered Planet," Cook, in "Human Fertility, the Modern Dilemma," Huxley, in "Evolution in Action," and Vogt, in "The Road to Survival," present the need of a biological interpretation of overpopulation.

It is plain to see that the main problems of man cannot be solved until populations are checked and *reduced*. Consider for a moment the effects of overpopulation on housing, on crowding, on cheapening and degradation of family life, on education in the home, in the primary and secondary schools and in the universities; consider the plundering of arable land, of natural resources, of wild life, of natural beauties; the international difficulties arising from population pressures, immigration, displaced persons, point-four program, etc.

John Stuart Mill was of the following opinion: "The density of population necessary to enable mankind to obtain in the highest degree, all of the advantages both of cooperation and of social intercourse has, in all the most populous countries, been attained. A population may be too crowded, though all be amply supplied with food and raiment. It is not good for man to be kept perforce at all times in the presence of his species. A world from which solitude is extirpated is a very poor ideal."

Why, may we ask, is society so indifferent to this vital problem? Like all man-made problems, the answer must be sought in the nature of man. Ernest A. Hooton, an outstanding authority in anthropology, discussed this very point in a recent address before the Case Institute of Technology at its diamond jubilee. Man, according to Hooton, "is still a superape; savage, predatory, acquisitive, primarily interested in himself."

A more optimistic note, however, was sounded in the superb advances of technology presented at the diamond jubilee. One paper described an electronic device which could play "a tolerably good game of checkers."

But in Hooton's opinion, the nature of man overshadows all shallow optimisms of technology. "The only possibility of improving the utilization of machines," in the words of Hooton, "lies in the improvement of man himself."

An improvement of man was proposed by applying to the human species the breeding methods man has devised to improve his domestic animals. Hooton, however, added that, "an immediate solution of human difficulties by turning out better breeds of man," is not in sight.

A concerted effort on the part of society to bring about greater humaneness might possibly meet the grave situation which faces it. It may, in fact, be the *only* road to salvation. In this effort, Medicine, whose sworn oath is to humanity, becomes a logical leader. And when Medicine broadens its point of view it will be best fitted to advise. It must come to appreciate the sweeping forces of evolution. It must come to recognize the fact that it is the normal healthy state of man, and not disease, that poses the major problems of civilization. It must find ways of meeting its responsibilities to society.

So allow me to end this talk, as I do each year in a lecture to the students in physiology on "The Biological Aspects of Medicine."

The medical profession possesses greater potential influence over the destiny of man than that of any other profession. The great numbers of the physician, his presence in all communities, the intimacy of his contacts, give him endless opportunities. But he must take the Oath of Hippocrates to mankind as conscientiously as he takes the Oath of Hippocrates to the individual.

—Excerpts from the Russell Lecture by Robert Gesell

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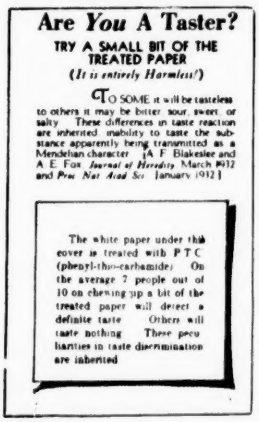
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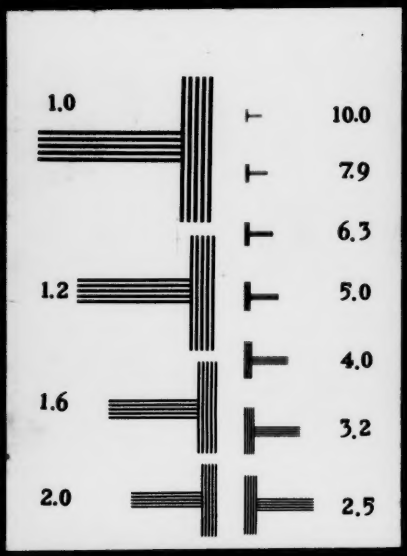

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